EXHIBIT 3

Declaration of Michael Weinberger

I, Michael Weinberger, M.D., declare under penalty of perjury the following to be true and accurate to the best of my information and belief:

1. I am a board-certified anesthesiologist and a board-certified pain medicine specialist. I also am a graduate of, and am on faculty at, Columbia University. My Curriculum Vitae is attached as Exhibit A.
2. I served an expert report in this case (Exhibit B) and intend to offer opinions at trial beginning concerning the Oklahoma Department of Corrections 2020 Execution Protocol ("Protocol") (Exhibit C) and, among other things, that I disagree with the opinions provided by Defendants' medical doctors that midazolam is suitable for how it is used in the Protocol or that there is any sound scientific basis for their opinions to that effect. Midazolam is not used clinically for this purpose or anything similar to this purpose because it is not used and should not be used as a sole drug to induce and maintain anesthesia.
3. I additionally intend to offer opinions at trial that the Protocol creates a substantial risk of severe pain and suffering. This is due, among other things, to the fact that midazolam is unsuitable to protect the Oklahoma death-row prisoners from severe pain and suffering expected to be caused by the Protocol, and that the consciousness check is wholly inadequate as provided

for in the Protocol in the context of using midazolam as the sole "anesthetic" as required by the Protocol.

4. I have reviewed the declarations of Meghan LeFrancois, an Assistant Federal Public Defender in the Western District of Oklahoma, and Julie Gardner, and investigator in the same office, as well as a press report by Associated Press reporter, Sean Murphy, annexed to Ms. Gardner's declaration and of which Ms. Gardner as an observer confirmed its accuracy.

5. These eyewitness accounts provide corroborative evidence of my above-stated opinions, specifically that the Protocol puts the prisoners at substantial risk of severe pain and suffering.

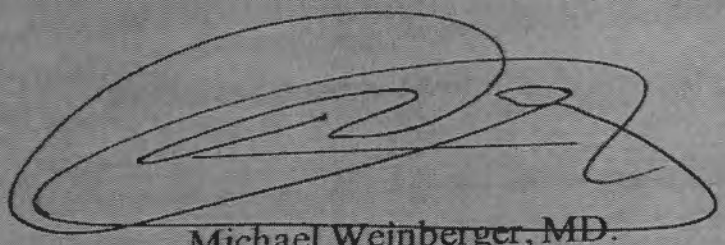
6. From the movements described in these reports by these witnesses, it would appear that these movements may have been voluntary and in view of these apparently voluntary movements of the head and the shoulder at about the time of the "consciousness check," the consciousness check as performed in this case was wholly inadequate.

7. The consciousness check described by the witnesses also appears inadequate to assess depth of sedation to assure the prisoner is insensate and under adequate general anesthesia before administering the second and third drugs which will cause substantial pain and suffering.

8. Consequently, it is reasonable to conclude that John Grant faced – after this inadequate consciousness check – a substantial risk of severe pain and suffering from the second and third drugs. Any other prisoners subjected to this Protocol administered in this manner would also be at substantial risk of severe pain and suffering as well.

/ / /

I declare under penalty of perjury the foregoing is true and accurate to the best of my belief.



Michael Weinberger, MD.

11/18/2021

Date

UNITED STATES DISTRICT COURT
WESTERN DISTRICT OF OKLAHOMA

RICHARD GLOSSIP, *et al.*,	)	
	)	
Plaintiffs,	)	
	)	
vs.	)	Case No. CIV-14-665-F
	)	
RANDY CHANDLER, *et al.*,	)	
	)	
Defendants.	)	

EXPERT OPINION OF DR. MICHAEL L. WEINBERGER

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I. EXPERIENCE AND EXPERT QUALIFICATIONS

1. I am currently the Medical Director of the Pain Management Center, Associate Professor of Anesthesiology at Columbia University Medical Center, and Section Chief, Pain Medicine at Columbia University Department of Anesthesiology in New York, New York. I have over 35 years of experience in anesthesiology and pain management. I am certified by the American Board of Internal Medicine; the American Board of Anesthesiology (Pain Medicine, Anesthesiology, Hospice and Palliative Medicine). My current *curriculum vitae* is attached as Exhibit A.

A. Summary of Education and Professional Experience

2. I graduated from Columbia University, College of Physicians and Surgeons, with an M.D. in 1983.

3. My post-graduate medical training included an internship in internal medicine at St. Vincent's Hospital and Medical Center, in New York, New York from 1983 to 1984. I was a Resident in internal medicine at St. Vincent's Hospital and Medical Center, in New York, New York from 1984 to 1986. I also served as a Resident in Anesthesiology at Columbia-Presbyterian Medical Center in New York, New York, from 1986 to 1989. And I completed a fellowship in Pain Management at Memorial Sloan Kettering Cancer Center in New York New York in 1990.

4. I am a member of numerous scientific and medical societies, including the Eastern Pain Association, the International Association for the Study of Pain, the American Society of Anesthesiologists, the American Society of Regional Anesthesia and Pain Medicine, the New York State Society of Anesthesiologists, and the Spinal Injection Society. I am currently on the Board of Directors Executive Committee of the Eastern Pain Association, and have previously served as its President. Additionally, I serve on the Guidelines Committee of the American Society of Anesthesiology and Regional Pain Medicine, and recently completed service on the Pain

Committee of the American Society of Anesthesiologists, as set out in my *curriculum vitae*, attached as Exhibit A.

5. I have given numerous lectures on anesthesiology and pain management as set out in detail in my *curriculum vitae*, attached as Exhibit A.

6. I am the author of several book chapters on anesthesiology and pain management. A full list of my publications is included in my *curriculum vitae*, attached as Exhibit A.

B. Prior Expert Testimony

7. I have served as a testifying expert in the following matters over the last four years:

- *Collegium Pharma. v. Teva Pharms.*, Case No. 1:18-cv-300 (D. Del.) 2019.
- *Janssen Pharms., Inc., et al. v. Actavis*, (D.N.J.) 2016.
- *Pernix Ireland Pain DAC and Pernix Therapeutics, LLC v. Alvogen Malta Operations LTD*, No 16-139 (D. Del.) 2018.
- *Valeant Pharms. Int'l Inc., et al. v. Mylan Pharms. Inc., et al.*, Case No. 15-8180 (D.N.J.) 2018 .

C. Compensation

8. I am being compensated at an hourly rate of $250 per hour. My compensation is not related in any way to opinions that I have formed or to the outcome of this or any other litigation.

II. OKLAHOMA'S THREE-DRUG LETHAL INJECTION PROTOCOL

9. I understand from my review of Oklahoma's February 20, 2020, execution procedures, Policy No. OP-040301, Execution of Offenders Sentenced to Death (the "Execution Protocol"), that Oklahoma intends to use the following three-drug lethal injection protocol:

A. Three-Drug Protocol with Midazolam, Vecuronium Bromide and Potassium Chloride in the Following Amounts:

1. The First Drug: 500 mg Midazolam

10. Administration of two syringes of midazolam, each containing a dose of 250 milligrams midazolam, for a total dose of 500 milligrams of midazolam, followed by a 60 ml heparin/saline flush.[1]

11. If after five minutes, a member of the execution team, the IV Team Leader, "physically confirm[s] the inmate is unconscious by using all necessary and medically-appropriate methods," then "the director will order the remaining chemicals be dispensed."[2] If the inmate is determined to remain conscious, the director shall determine how to proceed, including "if deemed appropriate, the director may instruct the Special Operations Team to administer additional doses of the chemical(s) [i.e., an additional 500 milligrams of midazolam] followed by the heparin/saline flush."[3]

12. The protocol does not specify the amount of time between the administration of the second and third chemicals, if any, but testimony indicates they are given in rapid succession upon the order to dispense being given.[4]

2. The Second Drug: 100 mg Vecuronium Bromide

13. Administration of two syringes of vecuronium bromide, each containing a dose of 50 milligrams, for a total dose of 100 milligrams of vecuronium bromide, followed by a 60 ml heparin/saline flush.[5] The protocol does not specify the volume of the syringes used to inject each

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[1] Execution Protocol, Attachment D, at ¶ C.4.a and Chart D.

[2] *Id.* at ¶¶ H.3 & H.4.

[3] *Id.* at ¶¶ H.5 & H.6.

[4] *Id.* at ¶ H.4, H.8; 11/12/2020 Crow Dep. Tr. 160:15-161:24

[5] *Id.* at ¶ C.4.b and Chart D.

50-milligram dose; therefore, it is not clear what the concentration of the drug will be or how long it will take to push.

14. The protocol does not specify whether any time elapses after administration of the vecuronium bromide before administration of the potassium chloride.[6] Testimony indicates they are given in rapid succession.[7]

3. The Third Drug: 240 mEq Potassium Chloride

15. Administration of two syringes of potassium chloride, each containing a dose of 120 milliequivalents, for a total dose of 240 milliequivalents of potassium chloride, followed by a 60 ml heparin/saline flush.

III. ASSIGNMENT AND MATERIALS CONSIDERED

16. I have been asked by counsel for Plaintiffs to provide an opinion based on my education, medical and professional training, knowledge, and experience, and the materials I have considered listed below, whether prisoners executed in accordance with the Execution Protocol face a substantial risk of experiencing severe pain and suffering as a result of the procedures set forth in the Execution Protocol.

17. In connection with this opinion, I have considered the following:

- The expected clinical effects and physical sensations of midazolam, specifically its effects on consciousness, awareness, and pain, including whether midazolam has a known lethal dose, suppresses breathing, is a sedative, or is an analgesic (painkiller).
- Whether midazolam is clinically used alone to induce and maintain general anesthesia.
- A description of the variability of the effects of midazolam between individuals, and the bases for this variability, including individual genetic variation, pharmacokinetics (the way drugs are metabolized by the body), interaction with other drugs, and the effects of certain medical conditions.

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[6] *See id.*

[7] 11/12/20 Crow Dep. Tr. 160:15-161:24.

- Whether midazolam has a "ceiling effect" such that after a certain dose, any increase in the amount administered will have no additional effect.

- Whether midazolam renders and maintains a subject in an insensate state and induces and maintains general anesthesia, even in the presence of severe noxious stimuli, such as intense pain, a sensation of suffocation, or paralysis.

- Whether this protocol, including midazolam, at the dosage and concentration used in the protocol, is likely to result in "flash" pulmonary edema, a painful medical complication in which fluid floods the lungs, causing sensations of air hunger, drowning, and/or suffocation. If so, whether midazolam renders and maintains a subject in an insensate state and induces and maintains general anesthesia during flash pulmonary edema.

- The expected clinical effects and physical sensations resulting from administration of the second and third drugs in Chart D of Attachment D of the Execution Protocol, vecuronium bromide and potassium chloride.

- Whether midazolam renders and maintains a subject in an insensate state and induces and maintains general anesthesia during the noxious stimuli and/or suffering caused by vecuronium bromide.

- Whether midazolam renders and maintains a subject in an insensate state and induces and maintains general anesthesia during the noxious stimuli of pain and suffering caused by potassium chloride.

- Whether the "consciousness check," method described in Attachment D, Paragraph H.3 of the Execution Protocol, is appropriate to ensure a subject is insensate to the noxious stimuli of pulmonary edema, the effects of vecuronium bromide, and the severe pain of administration of potassium chloride.

- Whether other "consciousness" checks and consciousness monitoring methods in Attachment D, including in Paragraphs H.8 and H.11 are appropriate to ensure a subject is, and through the execution continues to be, insensate to the noxious stimuli of pulmonary edema, the effects of vecuronium bromide, and the severe pain of the administration of potassium chloride.

- The expected clinical effect and physical sensations that would result from administration of the drugs listed in Chemical Chart D in Attachment D of the Execution Protocol without administration of the second drug listed in Chemical Chart D, vecuronium bromide.

- Whether other provisions of the Execution Protocol, including the personnel requirements, the training requirements, the intravenous ("IV") site preparation and establishment provisions, or the equipment provisions, increase the likelihood that a prisoner faces a substantial risk of experiencing severe pain and suffering.

- Whether additional procedural, training, or equipment requirements would potentially mitigate the risk that a prisoner executed in accordance with the Execution Protocol the likelihood that a prisoner faces a substantial risk of experiencing severe pain and suffering.

18. As a Board-Certified Anesthesiologist, I am prohibited from providing an opinion on whether one drug is more suitable for lethal injection than another. *See* Exhibit C. Accordingly, I was not asked by counsel for Plaintiffs to provide an opinion regarding whether any one of the drugs listed in Chemical Charts A, B, and D in Attachment D to the Execution Protocol, administered either individually or in combination with the other drugs listed in the Execution Protocol, or any other drugs not listed in the Execution Protocol, would be better execution drugs or more or less likely to cause prisoners executed by ODOC to face a risk of experiencing serious harm and severe pain and suffering. My opinions are limited to the inadequacies of the three-drug protocol.

19. The materials I have considered in forming my opinions are listed here:

- The February 20, 2020, Oklahoma Department of Corrections document OP-040301, titled "Execution of Inmates Sentenced to Death," referred to herein as the "Execution Protocol".
- Autopsy Reports of prisoners executed by lethal injection using midazolam in Alabama, Arkansas, Florida, Ohio, Oklahoma, Tennessee, and Virginia, included in Exhibit B.
- Press releases describing eye-witness accounts of executions of prisoners including the use of midazolam, as detailed in Exhibit B.
- Scientific and other publications and references, which are cited throughout this report and/or included in Exhibit B hereto.
- The expert report of Dr. Edgar in this matter.
- The Third Amended Complaint in this matter.

IV. SUMMARY OF OPINIONS

20. I provide here a summary of my opinions, each of which is described in further detail below, and each of which are based on my professional education, training, knowledge, and experience, and my review of the materials listed above. Unless indicated otherwise, each of my opinions is given to a reasonable degree of medical and scientific certainty.

21. It is my opinion that prisoners executed in accordance with Oklahoma's Execution Protocol, OSP Policy No. OP-040301, face a substantial risk of experiencing severe pain and suffering as a result of the Protocol's execution drugs and procedures.

22. It is also my opinion that midazolam, administered alone, cannot reliably render and maintain a subject in an insensate state and induce and maintain anesthesia, or block nociception (perception or transmission of a painful stimulus). It is widely accepted that midazolam, a sedative, should not be used as the only drug to induce and maintain anesthesia or render a patient insensate to pain.[8] If midazolam is used as an induction agent, it is typically used in combination with other drugs for rendering a patient insensate and for inducing anesthesia.[9] It is neither approved nor used for maintaining general anesthesia without other drugs.[10]

23. It is my further opinion that there is significant individual variation in response to midazolam, which can require a significant range in dosing to achieve similar results between individuals.[11] This variation is based on a number of factors, including, individual genetic

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[8] *E.g.*, Package Insert, Pfizer, Midazolam Injection, USP, at Indications and Usage.

[9] E.g., *id.*

[10] *E.g.*, *id.*; Amrein & Hetzel, *Pharmacology of Dormicum® (midazolam) and Anexate® (flumazenil)*, Acta Anasesthesiol Scand 1990: 34, Supplementum 92:6-15, at 13.

[11] *See, e.g.*, *id.* at Warnings ("Midazolam must never be used without individualization of dosage."); *E.g.*, Amrein & Hetzel, *supra* at 8 ("patients may react differently to equal doses of a given BZD or ***vice versa*** different doses may be needed to achieve the same effect in different patients").

differences, pharmacokinetics (the way the body metabolizes a drug), interaction with other drugs, and existing medical conditions.[12] This is one reason why midazolam is, as a general matter, not appropriate for use as a sole induction agent for general anesthesia and is not used as a sole agent for maintenance of general anesthesia.

24. It is also my opinion that midazolam is commonly referred to and understood as having a "ceiling effect."[13] This means that independent of any individual variation in responsiveness, additional amounts of the drug, even at much higher doses, will not produce an equally greater response in a subject or have a proportionally greater effect.

25. It is my further opinion that midazolam does not reliably render and maintain a subject in an insensate state and induce and maintain general anesthesia, or block nociception, in the presence of severe noxious stimuli, such as intense pain, or a sensation of suffocation or paralysis.

26. It is also my opinion that the three-drug protocol, including midazolam, in the dose and concentration called for in the protocol, leads to flash pulmonary edema, a condition involving severe suffering.[14] During flash pulmonary edema fluid floods the lungs, causing sensations of air hunger, drowning, and/or suffocation. Because midazolam does not reliably render a subject insensate, and maintain general anesthesia, or block nociception, in the presence of severe noxious stimuli, it is my further opinion that midazolam cannot reliably render and maintain a subject in an insensate state during the severe suffering caused by flash pulmonary edema.

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[12] *E.g.*, Amrein & Hetzel, *supra* at 8.

[13] *E.g.*, Amrein & Hetzel, *supra* at 6 ("a ceiling effect is observed after maximal doses of midazolam").

[14] See, e.g., Ikram, et al., Intravascular infusion of acid promotes intrapulmonary inducible nitric oxide synthase activity and impairs blood oxygenation in rats. Critical Care Medicine 2003; 31: 1454-1460 and Am J Respir Crit Care Med 1999: 159: 397-402.

27. It is my further opinion that midazolam is unlikely to render and maintain a subject in an insensate state and induce and maintain general anesthesia in the presence of the pain or suffering caused by vecuronium bromide and potassium chloride, the second and third drugs in ODOC's three-drug protocol, following midazolam administration. In particular, vecuronium bromide administered alone results in a slow death by asphyxiation caused by paralysis, which prevents a subject's ability to communicate and move. Potassium chloride administered alone produces cardiac arrest and a sensation of burning and intense pain as it circulates through the body. Midazolam is unlikely to render and maintain a subject in an insensate state and will not maintain general anesthesia, or block nociception, in the presence of the noxious stimuli caused by the effects of vecuronium bromide and the severe pain caused by potassium chloride.

28. It is my opinion that the "consciousness check" method described in Paragraphs H.3 and H.8 of Attachment D to the Execution Protocol is not sufficient to ensure a subject is insensate to the noxious stimuli of pulmonary edema, effects of vecuronium bromide, and potassium chloride.

29. It is my further opinion that if the first and third drugs in ODOC's three-drug protocol, midazolam and potassium chloride, were administered to a subject without the second drug in the three-drug protocol, vecuronium bromide, a subject would be able to communicate or otherwise demonstrate through physical or other responses, their experience of flash pulmonary edema as described above, including the sensations of shortness of breath and excruciating air hunger, similar to a sensation of drowning. The subject would also be able to communicate or otherwise demonstrate through physical or other responses, their experience of burning, and intense pain as the potassium chloride circulated through their body. This is based on my opinion above that midazolam administered alone is unlikely to render a subject insensate and maintain

unconsciousness in the presence of severe noxious stimuli, such as intense pain. Without administration of vecuronium bromide the subject would be able to communicate the sensations experienced or at a minimum show physical response resulting from administration of potassium chloride and the midazolam itself.

30. Additionally, it is my opinion that the personnel requirements, training requirements, "consciousness check" provisions, intravenous ("IV") site preparation and establishment provisions, and the equipment provisions of the Execution Protocol, as described below, create or contribute to a risk that a prisoner executed in accordance with the Execution Protocol will experience serious harm and severe pain and suffering. These requirements and provisions relate to medical decisions necessitated by the Execution Protocol procedures and potential medical outcomes that will result from the Execution Protocol procedures. Additional provisions or changes to the existing procedural, training, or equipment requirements would mitigate the risk that a prisoner executed in accordance with the Execution Protocol will experience serious harm and severe pain and suffering.

V. BACKGROUND AND OPINIONS

A. Drugs Used in Oklahoma's Three-Drug Midazolam Execution Protocol

1. Midazolam

31. The first drug in Oklahoma's three-drug lethal injection protocol is midazolam. Midazolam is a member of the benzodiazepine class of drugs, which are used primarily as sedative/hypnotic (sleep-producing) agents. Other benzodiazepines include diazepam (Valium®), and alprazolam (Xanax®).

32. Although benzodiazepines differ in some specific aspects of their properties, they are qualitatively very similar, and most benzodiazepines may be used interchangeably with appropriate adjustments to reflect differences in potency, solubility, bioavailability, and

duration of action.[15] "All benzodiazepines have hypnotic, sedative, anxiolytic, amnesic, anticonvulsant, and centrally produced muscle-relaxing properties. They may differ to some extent in their potency and efficacy with regard to some of these pharmacodynamic actions (e.g., anticonvulsive action)."[16]

33. All benzodiazepines share the same mechanism of action. Benzodiazepines bind to molecular components of the $GABA_A$ receptor in neuronal membranes (the outer wall of neuron cells) in the central nervous system. This receptor, depicted below, functions as a chloride ion channel, and is activated by the inhibitory neurotransmitter GABA.[17]

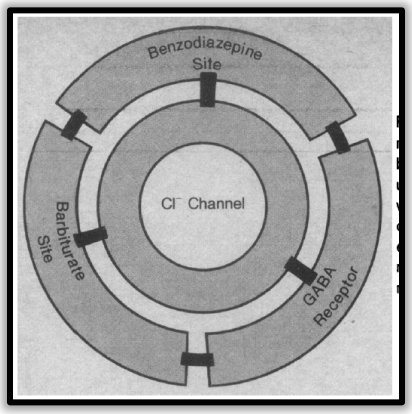

***Figure 1**: The $GABA_A$ receptor showing multiple binding sites and the chloride ion channel.*

[15] Mihic SJ, Mayfield J, Harris RA. *Hypnotics and Sedatives.* 2017. In Brunton LL, Hilal-Dandan R, Knollmann BC, editors. Goodman and Gillman's The Pharmacological Basis of Therapeutics. 13th ed. McGraw-Hill Education/Medical.

[16] Vuyk J, Sitsen E, Reekers M. 2019. Intravenous Anesthetics. In: Gropper M, Eriksson L, Fleisher L, Wiener-Kronish J, COhen N, Leslie K, editors. Miller's Anesthesia. 9th ed. Elsevier. pp. 638-679.

[17] Study, JAMA (1982); Mihic, *supra*.

34. Although many other drugs including barbiturates, and the "z" drugs (zolpidem or Ambien®, zaleplon, and eszopiclone), also bind the $GABA_A$ receptor, as shown above, their mechanism of action is distinct. Benzodiazepines, including midazolam, do not function in the absence of the GABA neurotransmitter, but instead enhance the natural effects of GABA, causing the chloride channels to open more frequently,[18] as depicted in the diagram below:

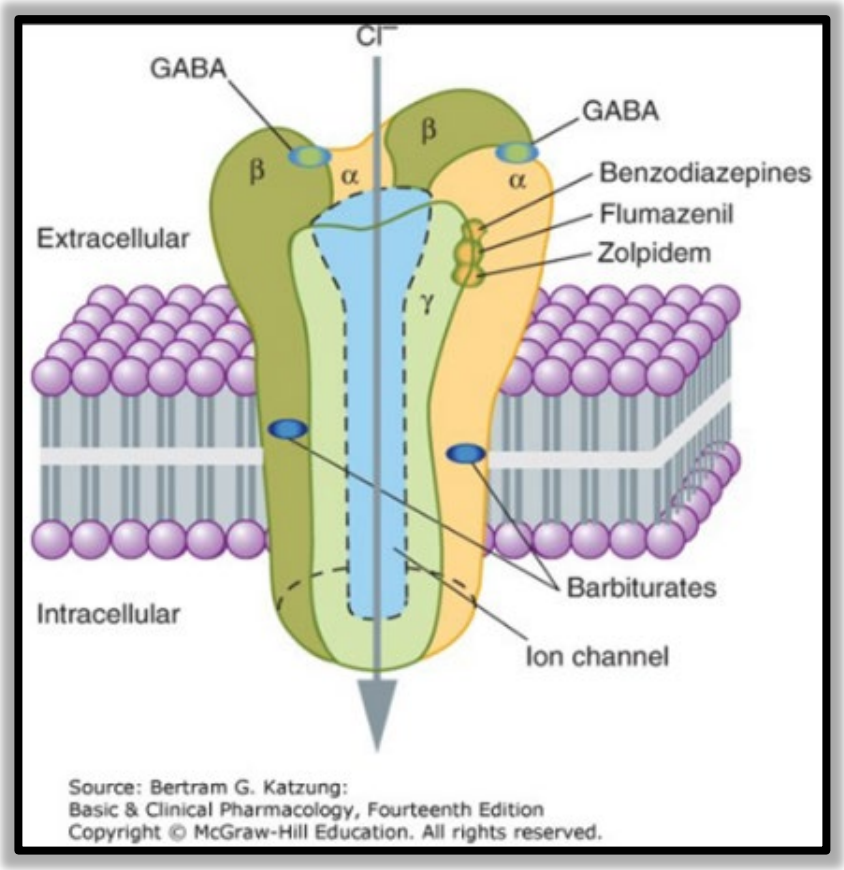

***Figure 2**: The $GABA_A$ receptor spanning the cellular membrane, with unique binding sites for the $GABA_A$ neurotransmitter molecule, benzodiazepines, and barbiturates.*

[18] Mihic, *supra*; Bai, et al., *Distinct Functional and Pharmacological Properties of Tonic and Quantal Inhibitory Postsynaptic Currents Mediated by γ-Aminobutyric AcidA Receptors in Hippocampal Neurons*, Molecular Pharmacology Vol. 59, No. 4, 814-824, 2001.

35. Barbiturates also enhance the effect of the GABA neurotransmitter, "but—in contrast to benzodiazepines—they increase the *duration* of the GABA-gated chloride channel openings."[19] And at high concentrations, barbiturates may also directly activate the chloride channels, even in the absence of the GABA neurotransmitter, by binding at other sites, which may be the basis for barbiturates' ability to induce full surgical anesthesia and for their overall lower margin of safety than the benzodiazepines such as midazolam.[20]

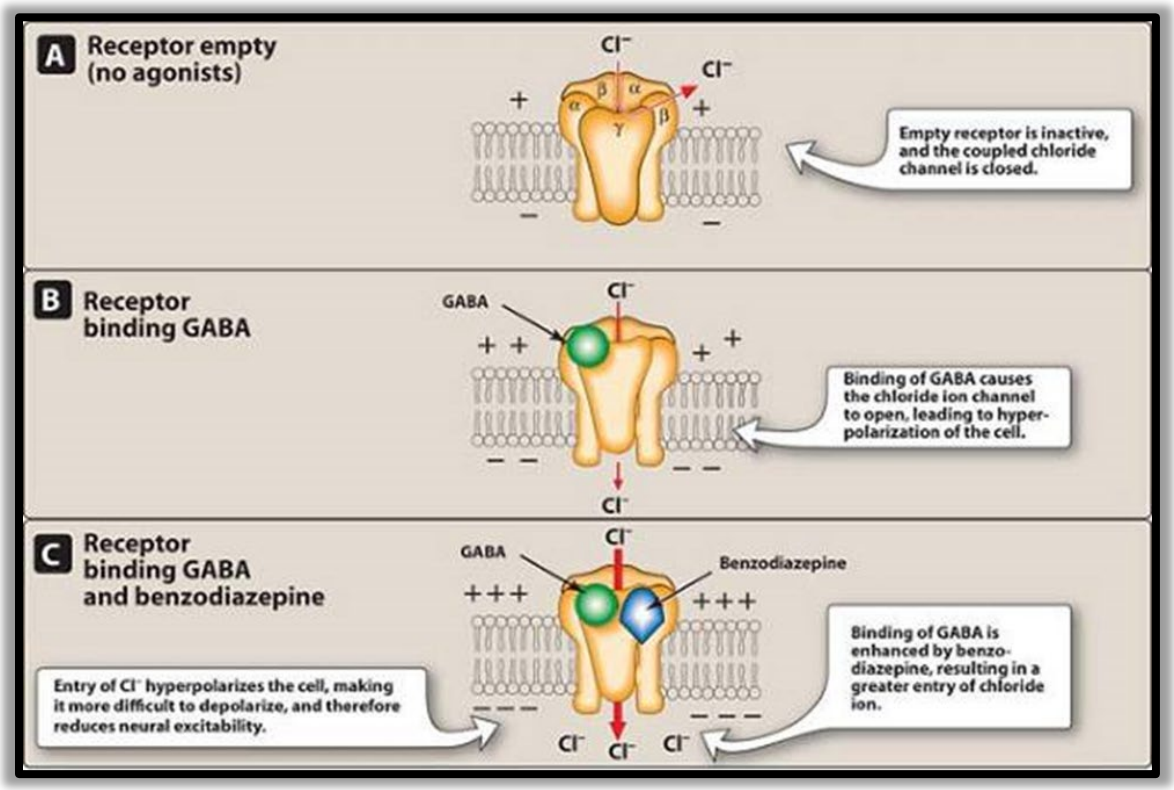

***Figure 3**: Benzodiazepines increase the frequency of the $GABA_A$ receptor ion opening, resulting in more movement of chloride channels across the cellular membrane. Benzodiazepines have no independent effect if GABA is not present.*

[19] Mihic, *supra*.

[20] Mihic, *supra*.

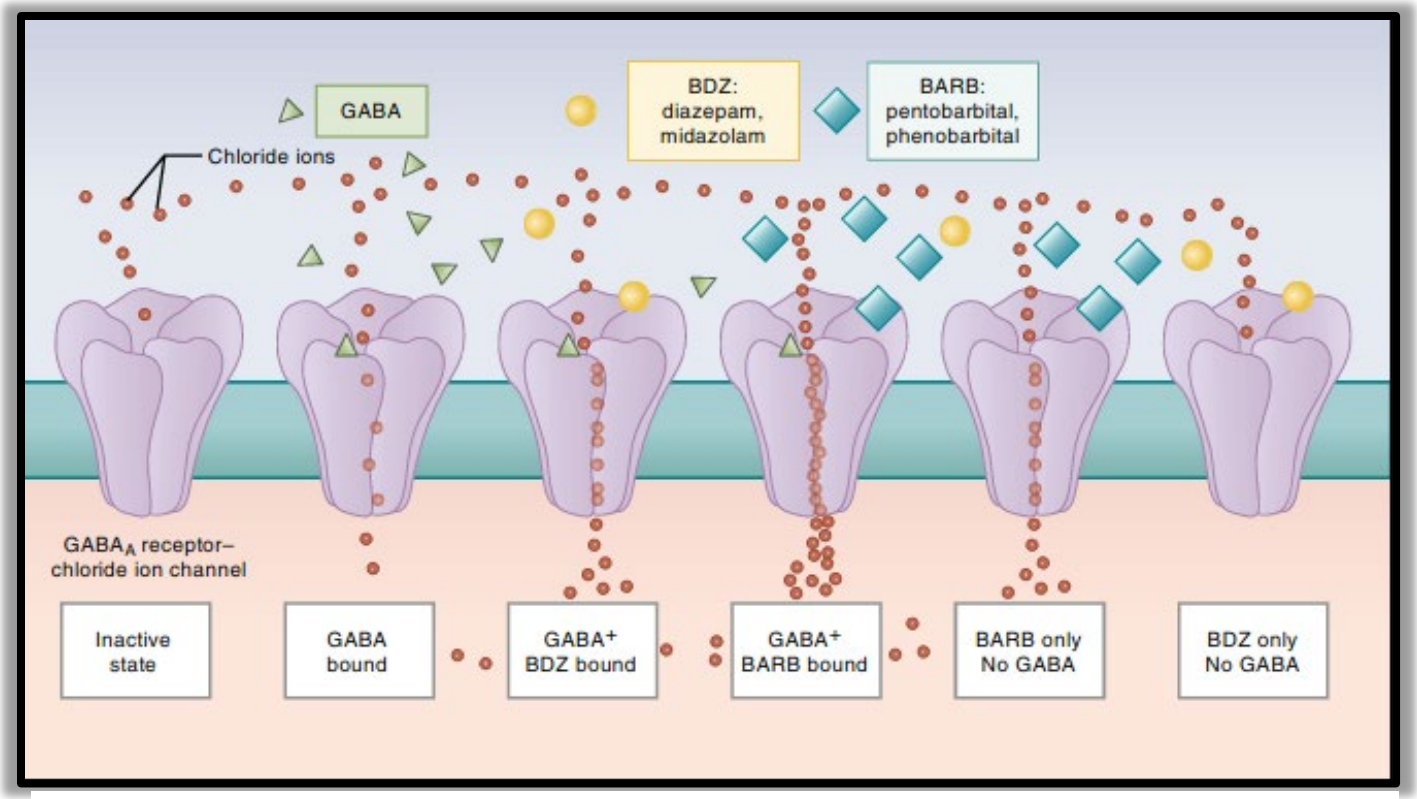

Figure 4: The GABA$_A$ Receptor shown with no binding; with binding of the GABA neurotransmitter only; with binding of GABA and a Benzodiazepine; with binding of GABA and a Barbiturate; with binding of a Barbituate only without the presence of GABA; and with binding of a Benzodiazepine only, without the presence of GABA.

36. As shown in Figure 4 above, the major difference between benzodiazepines, such as midazolam, and barbiturates, such as pentobarbital, is that in the absence of the naturally occurring GABA neurotransmitter, benzodiazepines have no effect on the GABA$_A$ receptor and therefore cannot exert an effect. In contrast, the barbiturates can exert an effect on the GABA$_A$ receptor even when the GABA neurotransmitter is not present.

37. Benzodiazepines, including midazolam, are routinely classified as hypnotics and sedatives. The most prominent effects of benzodiazepines are "sedation, hypnosis, decreased anxiety, muscle relaxation, anterograde amnesia, and anticonvulsant activity."[21]

[21] Mihic, *supra*; *see id.* at Table 19-2.

"The benzodiazepines have become a category of drugs widely used in anesthesia as anxiolytics, sedatives, and hypnotics. . . . In the clinical practice of daily anesthesia, midazolam is often used immediately before induction of anesthesia."[22]

38. "Although the clinical literature often refers to the 'anesthetic' effects and uses of certain benzodiazepines, these drugs do not cause a true general anesthesia; awareness usually persists, and a failure to respond to a noxious stimulus sufficient to allow surgery cannot be achieved."[23]

39. Midazolam, like other benzodiazepines, also lacks analgesic (pain-relieving) properties.[24]

40. Indeed, midazolam is approved by the FDA only for the following indications:[25]

Midazolam injection is indicated:

- Intramuscularly or intravenously for preoperative sedation/anxiolysis/amnesia;
- Intravenously as an agent for sedation/anxiolysis/amnesia prior to or during diagnostic, therapeutic or endoscopic procedures, such as bronchoscopy, gastroscopy, coronary angiography, cardiac catheterization, oncology procedures, radiologic procedures, suture of lacerations and other procedures either alone or in combination with other CNS depressants;
- Intravenously for induction of general anesthesia, before administration of other anesthetic agents. With the use of narcotic premedication, induction of anesthesia can be attained within a relatively narrow dose range and in a short period of time. Intravenous midazolam can also be used as a component of intravenous supplementation of nitrous oxide and oxygen (balanced anesthesia);

[22] Vuyk, et al., *Intravenous Anesthetics*, *in Miller's Anesthesia*, 9th Edition (Gropper, ed.).

[23] Mihic, *supra*. & Table 19-2; Vuyk, et al., *Intravenous Anesthetics*, *in Miller's Anesthesia*, 9th Edition (Gropper, ed.).

[24] *E.g.* Mihic, *supra*.

[25] Pfizer – IFU.

- Continuous intravenous infusion for sedation of intubated and mechanically ventilated patients as a component of anesthesia or during treatment in a critical care setting.[26]

41. Notably, midazolam is not FDA approved for maintenance of general anesthesia, or for pain relief. Instead it is approved for "induction of general anesthesia, *before administration of other anesthetic agents*."[27] All other approved indications are for preoperative sedation, conscious sedation, or require midazolam to be used as a component of anesthesia with other drugs.

42. Because midazolam is a sedative, rather than an anesthetic or analgesic, it is unlikely to produce general anesthesia in humans when used alone. And in my experience I have never seen it used as a sole drug for general anesthesia. It is used infrequently as a solo drug for moderate sedation and for "conscious sedation" in some low-discomfort procedures such as colonoscopy, which is of such low pain stimulus that it can be performed in the majority of patients without any sedation at all.[28] Used alone, however, midazolam cannot reliably produce general anesthesia, which is defined by the American Association of Anesthesiology as "a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation."

43. Midazolam alone is also usually not a lethal drug. While benzodiazepines such as midazolam may be listed as the "cause of death" in drug overdose cases, the actual "mode" of death is not due to direct drug toxicity (midazolam does not have a reported lethal toxic dose[29]). Potential mechanisms of death from benzodiazepine overdose are generally centered around

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[26] Pfizer – IFU.

[27] Pfizer – IFU (emphasis added).

[28] Hoffman MS, Butler TW, Shaver T. *Colonoscopy without sedation*. J Clin Gastroenterol 1998; 26:279-82; *see also* Pfizer – IFU – Indications and Usage.

[29] See Schultz M, Iwersen-BergmannS, Andresen H, Schmoldt A. *Therapeutic and toxic blood concentrations of nearly 1,000 drugs and other xenobiotics*. Crit Care 2012; 16:R136; *see also* Cheng, et al., *When Midazolam Fails, Journal of Pain and Symptom Management*, Vol. 23 No. 3 March 2002, at 261.

interruption of breathing that results in subsequent suffocation, due to muscle sedation. This can occur due to interference with the brain's regulation of breathing during benzodiazepine-induced sedation, particularly when combined with narcotics; mechanical airway obstruction from collapse of the tissues into the airway because of reduced muscle tone in the throat during benzodiazepine-induced sedation; aspiration of stomach contents into the airway during sedation ("drowning in vomit"); and other mechanisms.[30] Supportive measures (e.g. assisted ventilation) during large doses of midazolam ·prevent death, because midazolam itself does not have a reported lethal toxic dose.

44. The safety profile of midazolam in large doses is in contrast to the barbiturate class of drugs, which are known to commonly result in death from overdose. This is because barbiturates, in contrast to the benzodiazepines, depress both the respiratory drive and the mechanisms responsible for rhythmic breathing.[31]

45. At clinical doses, midazolam has no known direct deleterious effects on cells in the body, and produces no irreversible effects, but can temporarily interfere with the body's normal responses, such as breathing more deeply in response to hypoxemia (lack of oxygen) or hypercarbia (high levels of carbon dioxide in the blood). This is in contrast to drugs in which intrinsic lethal toxicity occurs, such as cyanide, which irreversibly stops oxidative function of cells. Even extremely high doses of benzodiazepines that have been administered or taken have not been fatal,[32] whereas even small doses of cyanide are usually lethal.

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[30] *E.g.*, Vuyk, et al., *supra*.

[31] Mihic, *supra*.

[32] *Id.*; *see also* Divoll M et al. (1981) *Benzodiazepine overdosage: plasma concentrations and clinical outcome*. Psychopharmacology (Berl) 73:381-383 (twelve patients who overdosed on a benzodiazepine alone (diazepam), in doses as high as 750 mg, suffered no ill effects, were minimally sedated, and discharged from the emergency department within 24 hours); Allen MD

46. In addition, the effects of a single dose of midazolam may vary significantly between individuals due to numerous factors including genetic variation, pharmacokinetics (how quickly the drug is metabolized), the method of administration of the drug; the individual to whom it is administered,[33] and any stimulus applied or experienced during its administration.

47. The dose of midazolam required to produce sedation is also dependent on prior or concomitant use of other medications. Midazolam "undergoes biotransformation by the P450 system," which is conducted primarily in the liver.[34] Concomitant use of drugs that stimulate, or induce, the P450 system may cause benzodiazepines, including midazolam, to be metabolized, or broken down more rapidly.[35] This may cause a reduced onset of action of midazolam or shorten or augment its effect and duration of action. Prior or concomitant illicit drug use by the recipient may also reduce the effects of the drug due to cross-tolerance (thus requiring higher than expected doses to produce a desired effect), or alternatively can augment the effects of the drug through additive action.[36]

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et al. (1980) *Pharmacokinetic study of lorazepam overdosage*. Am J Psychiatry 137:1414-1415 (patients who overdosed on another benzodiazepine, lorazepam, were admitted to the ER with blood levels 12 times greater than therapeutic concentrations; all responded to a light pain stimulus); Greenblatt DJ et al. (1977) *Acute overdosage with benzodiazepine derivatives.* Clin Pharmacol Ther. 21497-514 (of twelve benzodiazepine overdose patients with no other drugs in their system, none needed assisted ventilation and all were discharged within two days with no ill effects; these included doses of 2.5 grams of chlordiazepoxide (Librium®) and 400 mg of diazepam (Valium®)).

[33] *See, e.g.*, *id.* at Warnings ("Midazolam must never be used without individualization of dosage."); *E.g.*, Amrein & Hetzel, *supra* ("patients may react differently to equal doses of a given BZD or ***vice versa*** different doses may be needed to achieve the same effect in different patients").

[34] Cheng, *supra*.

[35] E.g., Vuyk, et al., *supra*, at 657.

[36] *See* Vuyk, et al., *supra*, at 657; Mihic, *supra*.

48. In sum, midazolam is a benzodiazepine that is approved for and widely used as a preoperative sedative or hypnotic, prior to administration or in conjunction with anesthetic drugs, or for conscious sedation during low pain procedures such as colonoscopy. It is not capable of maintaining general anesthesia, as that term is defined by the American Association of Anesthesiologists and is almost never used to do so. It has no analgesic, or pain-relieving properties,[37] and in fact may at times increase the experience of pain.[38] Its effects at the individual level are highly variable due to genetics, administration route, dosage, and drug-drug interaction, among other things. It is not reported to have a known lethal dose, and is considered relatively safe, even at high doses.

2. Vecuronium Bromide

49. The second drug in Oklahoma's three-drug lethal injection protocol is vecuronium bromide. Vecuronium bromide is a nondepolarizing neuromuscular blocking agent. Other nondepolarizing neuromuscular blocking agents include pancuronium, and rocuronium.[39] Neuromuscular blocking agents are typically used as anesthetic adjuncts in order "to relax muscles of the jaw, neck, and airway and thereby facilitate laryngoscopy and endotracheal intubation."[40]

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[37] "Benzodiazepines lack analgesic properties and must be used with other anesthetic drugs to provide sufficient analgesia." Vuyk, et al., *Chapter 30: Intravenous Anesthetics*, Miller's Anesthesia (2015) Eighth Edition. (pp. 821-863), Elsevier: Saunders, Philadelphia, PA.

[38] "Midazolam increased cold, heat and electrical pain perception significantly." Frölich MA et al. (2013) *Effect of sedation on pain perception*. Anesthesiol. 118:611-621; Prosenz J, Gustorff B (2017) *Midazolam as an active placebo in 3 fentanyl-validated nociceptive pain models*. Pain 158:1264-1271.

[39] *See, e.g.*, Pfizer-IFU- uspi_vecuronium.pdf (pfe-pfizercom-d8-prod.s3.amazonaws.com)

[40] Patel, Hemal H., et al. "General Anesthetics and Therapeutic Gases." *Goodman & Gilman's: The Pharmacological Basis of Therapeutics, 13e* Eds. Laurence L. Brunton, et al. McGraw-Hill, 2017, available at https://accessmedicine.mhmedical.com/content.aspx?bookid=2189&sectionid=170269975 (last visited Dec. 29, 2020); Pfizer-IFU- uspi_vecuronium.pdf (pfe-pfizercom-d8-prod.s3.amazonaws.com), at Indications and Usage.

50. A typical clinical dose is between 0.08 and 0.1 mg/kg, or for a 200 lb person, approximately 9 mg: "An initial vecuronium bromide dose of 0.08 to 0.1 mg/kg generally produces first depression of twitch in approximately 1 minute, good or excellent intubation conditions within 2.5 to 3 minutes, and maximum neuromuscular blockade within 3 to 5 minutes of injection in most patients."[41] "The time to onset of paralysis decreases and the duration of maximum effect increases with increasing vecuronium doses."[42] Time to recovery after an initial dose of 0.08 to 0.1 mg/kg is on average 15-25 minutes. The dose called for by Oklahoma's execution protocol is approximately 10 times the dose administered in a clinical setting.

51. Vecuronium acts by competing for cholinergic receptors on motor endplates, the junction between a motor nerve fiber and a muscle fiber, thereby inhibiting depolarization of muscle neurons.[43]

52. Vecuronium, like all paralytic agents, has no analgesic or sedative properties of its own, and does not have any effect on consciousness.[44] As a result, a subject who receives vecuronium without concurrent sufficient sedation or anesthetic, is conscious and able to feel pain,

[41] Pfizer-IFU- uspi_vecuronium.pdf (pfe-pfizercom-d8-prod.s3.amazonaws.com)

[42] Pfizer-IFU- uspi_vecuronium.pdf (pfe-pfizercom-d8-prod.s3.amazonaws.com), at Clinical Pharmacology; *see also* Sharpe MD, Murking JM, Vannelli T. *High-dose vecuronium neuromuscular block: a comparison of arrhythmias and onset of block during sufentanil anaesthesia*. Can J Anaesth 1995; 42:716-23.

[43] Vecuronium, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com).

[44] *E.g.*, Pfizer-IFU- uspi_vecuronium.pdf (pfe-pfizercom-d8-prod.s3.amazonaws.com), at C.N.S. ("Vecuronium has no known effect on consciousness, the pain threshold or cerebration. Administration must be accompanied by adequate anesthesia or sedation."); Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com).

but unable to communicate in any way.[45] When this occurs as a result of anesthetic error, the experience is so distressing that it can result in post-traumatic stress disorder.[46]

3. Potassium Chloride

53. Potassium chloride (or KCl) is the third drug in Oklahoma's three-injection protocol. KCl is a naturally occurring mineral salt that can be administered orally or intravenously. Potassium ions are required for a number of important functions in the human body, including "conduction of nerve impulses in heart, brain, and skeletal muscle; contraction of cardiac, skeletal and smooth muscles; maintenance of normal renal [kidney] function, acid-base balance, carbohydrate metabolism, and gastric secretion."[47]

54. Potassium chloride for injection is typically used to treat hypokalemia, a condition in which blood potassium levels are low, which can lead to muscular paralysis, respiratory failure, and cardiac abnormalities.[48] Potassium chloride has also been used historically during cardiac surgery to stop the heart during heart bypass surgery.

55. Injection of KCl without sufficient doses of an appropriate anesthetic or analgesic can cause severe pain, and therefore in medical practice KCl is only given in very dilute concentrations, and very slowly. High concentrations of potassium chloride can cause severe pain because the potassium ions depolarize, or activate, all of the sensory nerves, including pain nerves,

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[45] *See, e.g.*, Mashour GA. Posttraumatic stress disorder after intraoperative awareness and high-risk surgery. Anesth Analg 2010; 110: 668–70; Avidan, et al., Prevention of Intraoperative Awareness in a High-Risk Surgical Population. N Engl J Med 2011; 365:591-600.

[46] *See, e.g.*, Mashour GA. Posttraumatic stress disorder after intraoperative awareness and high-risk surgery. Anesth Analg 2010; 110: 668–70; Avidan, et al., Prevention of Intraoperative Awareness in a High-Risk Surgical Population. N Engl J Med 2011; 365:591-600.

[47] Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com)

[48] Kruse JA, Carlson RW (1990) Rapid correction of hypokalemia using concentrated intravenous potassium chloride infusions. Arch Intern Med. 150:613-617. Potassium Chloride, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com).

in the veins.[49] As a result, The Institute for Safe Medication Practices (ISMP) includes this medication (IV formulation) among its list of drugs which have a heightened risk of causing significant patient harm when used in error.[50]

56. When potassium chloride is administered to treat hypokalemia it is administered slowly in dilution.[51] This is because even such a small dose-which is not likely to produce heart arrhythmias-when diluted in small volumes of fluid (< 1 liter), results in a high enough concentration of KCl that the drug damages tissues, causing severe pain.[52]

57. Concentrations of more than 80-100 mEq/L (milliequivalents per liter)[53] are known to cause severe pain.[54] Some clinicians therefore recommend that the maximum concentration

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[49] Parsons CL (2011) The role of a leaky epithelium and potassium in the generation of bladder symptoms in interstitial cystitis/overactive bladder, urethral syndrome, prostatitis and gynaecological chronic pelvic pain. BJU Int. 107:370-375; Williams RH (1973) *Potassium overdosage: a potential hazard of non-rigid parenteral fluid containers*. Br Med J. 1:714-715.

[50] Potassium Chloride, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com).

[51] Potassium Chloride, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com).

[52] *See* Parsons CL (2011) *The role of a leaky epithelium and potassium in the generation of bladder symptoms in interstitial cystitis/overactive bladder, urethral syndrome, prostatitis and gynaecological chronic pelvic pain*. BJU Int. 107:370-375; Lankton JW et al. (1973) Letter: *Hyperkalemia after administration of potassium from nonrigid parenteral-fluid containers*. Anesthesiology 39:660-661; Williams RH (1973) *Potassium overdosage: a potential hazard of non-rigid parenteral fluid containers*. Br Med J. 1:714-715.

[53] The convention for dosage of mineral salts such as potassium chloride is in mEq, or in mEq per liter of whatever fluid in which they are diluted. A physician must always not only designate the total dose (mEq), but how to dilute the salt (mEq/L) because both affect the clinical effects. For context, a common dose range of potassium chloride (KCl) is 25 to 40 mEq, delivered in 1 liter of fluid (i.e. a concentration of25 to 40 mEq/L) over 8 to 10 hours. Pucino F, et al, *Patient tolerance to intravenous potassium chloride with and without lidocaine*. Drug Intell Clin Pharm 1988; 22:676-9.

[54] Pucino, et al., *supra*; Parsons, *supra*.

for peripheral infusion not exceed 10mEq/mL. The maximum rate of administration for peripheral infusion is 10 mEq/hour.[55]

58. Rapid intravenous administration of KCl is used in lethal injection protocols to cause rapid onset of a fatal heart arrhythmia, i.e., to "stop the heart".[56] The Oklahoma Execution Protocol calls for a dose of two injections of 120 mEq of KCl for a total dose of 240 mEq, without any indication of the volume in which it is diluted. The testimony indicates that the potassium is administered as quickly as possible. If the dose is delivered in two 60 cc syringes,[57] this would result in administration of a dose of potassium chloride that is significantly greater than doses known to cause severe pain.[58] In a patient not under general anesthesia, the dose called for in the protocol would cause severe pain.

B. Consciousness, Pain, Noxious or Nociceptive Stimuli and Anesthetic Induction, Maintenance, Monitoring, and Depths

1. Consciousness Is Not Determinative of the Elimination of Pain and Suffering

59. Consciousness is not considered determinative of whether or not an individual is experiencing pain or suffering. Consciousness is one of several components used to determine whether an individual is in a state of general anesthesia: "General anesthesia is a drug-induced reversible condition composed of four behavioral and physiological states: antinociception,

[55] Potassium Chloride, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com)

[56] 11/12/20 Crow Dep. Tr. At 102:20.

[57] OAG 017824-017828.

[58] Pucino F, Danielson BD, Carlson JD. *Patient tolerance to intravenous potassium chloride with and without lidocaine*. Drug Intell Clin Pharm 1988; 22:676-9.

unconsciousness, amnesia, [and] immobility; and stability of the physiologic systems, including the autonomic, cardiovascular, respiratory, and thermoregulatory systems."[59]

60. General anesthesia is thus defined not only as loss of consciousness, but as "a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation."[60] But a lack of arousal, or consciousness, alone is considered insufficient to determine whether pain and suffering has been eliminated. For example, individuals with consciousness disorders, such as a brain injuries or individuals in a vegetative or minimally conscious state, may be unconscious and unable to communicate, but are assessed using numerous mechanisms to determine their ability to experience pain and suffering.[61] For example, in addition to motor and language responses, patients considered "unconscious" or "minimally conscious," children too young to communicate pain verbally, elderly patients suffering from dementia, or sedated/intubated patients are assessed by monitoring facial expression, vocalization other than language, body movements, physiological parameters such as heart rate, breathing patterns, or blood pressure, and changes in activity.[62]

61. Therefore, instead of relying on a determination of "consciousness," anesthesiologists rely on the concept of antinociception to measure adequacy of anesthesia and perception of pain and suffering. Nociception refers to the transmission of potentially harmful and

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[59] Brown, et al., *Monitoring the State of the Brain and Central Nervous System During General Anesthesia and Sedation, in Miller's Anesthesia*, 9th Edition (Gropper, ed.).

[60] *E.g.*, Brown, et al., *Monitoring the State of the Brain*, *supra*; Schankers C, Chatelle C, Demertzi A, Majerus S, Laureys S. What about pain in disorders of consciousness? The AAPS Journal 2012;14(3):437-444.

[61] Schnakers, et al., *supra.*

[62] Schnakers, et al., *supra*.

noxious stimuli through the sensory system. Antinociception refers to "the action or process of blocking the detection of a painful or injurious stimulus by sensory neurons."[63]

2. Pain, Suffering, and Nociception

62. Pain and suffering are the result of a neurobiological system referred to collectively as nociception, or the nociceptive (pain) pathway.[64] The nociceptive system, or circuit, is illustrated in the figure below:

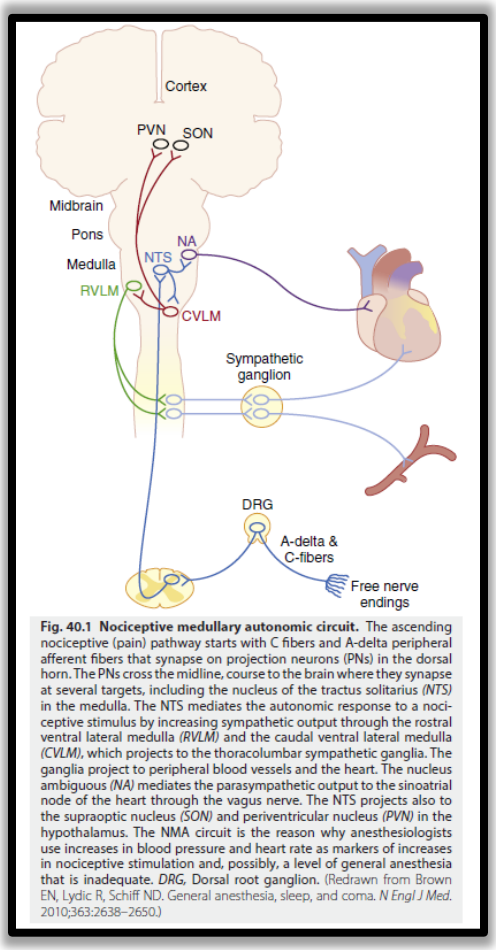

Fig. 40.1 Nociceptive medullary autonomic circuit. The ascending nociceptive (pain) pathway starts with C fibers and A-delta peripheral afferent fibers that synapse on projection neurons (PNs) in the dorsal horn. The PNs cross the midline, course to the brain where they synapse at several targets, including the nucleus of the tractus solitarius *(NTS)* in the medulla. The NTS mediates the autonomic response to a nociceptive stimulus by increasing sympathetic output through the rostral ventral lateral medulla *(RVLM)* and the caudal ventral lateral medulla *(CVLM)*, which projects to the thoracolumbar sympathetic ganglia. The ganglia project to peripheral blood vessels and the heart. The nucleus ambiguous *(NA)* mediates the parasympathetic output to the sinoatrial node of the heart through the vagus nerve. The NTS projects also to the supraoptic nucleus *(SON)* and periventricular nucleus *(PVN)* in the hypothalamus. The NMA circuit is the reason why anesthesiologists use increases in blood pressure and heart rate as markers of increases in nociceptive stimulation and, possibly, a level of general anesthesia that is inadequate. *DRG*, Dorsal root ganglion. (Redrawn from Brown EN, Lydic R, Schiff ND. General anesthesia, sleep, and coma. *N Engl J Med.* 2010;363:2638–2650.)

[63] Merriam-Webster, Antinociception Medical Definition | Merriam-Webster Medical Dictionary (merriam-webster.com); Brown, et al., *Monitoring the State of the Brain*, *supra.*

[64] *E.g.*, Hurley, et al., *Acute Postoperative Pain*, *in Miller's Anesthesia*, 9th Edition (Gropper, ed.); Brown, et al., *Monitoring the State of the Brain*, *supra*.

63. The nociceptive, or pain pathway, begins with nerve endings that carry nociceptive, or pain, information from the peripheral nervous system to the spinal cord.[65] In response to stimuli indicating potential or actual tissue injury, the nociceptors, or pain receptors, first transmit information to the spinal cord. In the spinal cord, that information is further processed and transmitted to the brain stem. In the brain stem, the signal is further processed and transmitted and can produce an almost-instantaneous autonomic response, such as changes in heart rate, respiratory rate, and blood pressure.[66] The autonomic system is often referred to as the system responsible for the "fight-or-flight" response.

64. This nociceptive system is central to understanding how pain and suffering is perceived, and how an individual's awareness and perception of pain and suffering is assessed by anesthesiologists.[67] The nociceptive system is "the pathway most used by anesthesiologists for monitoring [a] patient's level of unconsciousness and antinociception."

65. The activity in the nociceptive, or pain, pathway is thus rapidly observed when a patient is under general anesthesia "because this pathway is a fundamental component of the fight-or-flight response." While nociceptive information may also be further transmitted to higher centers of the central nervous system where it may induce responses in the brain cortex, the nociceptive circuit and the sympathetic responses it produces is "used as a sentinel for detecting nociceptive stimuli that can lead to autonomic, stress, and arousal responses."[68] Because motor responses may be blocked by muscle relaxation, changes in heart rate and blood pressure in

[65] Brown, et al., *Monitoring the State of the Brain*, *supra*; Hurley, et al., *supra*.

[66] Brown, et al., *Monitoring the State of the Brain*, *supra*;

[67] Brown, et al., *Monitoring the State of the Brain*, *supra*.

[68] Brown, et al., *Monitoring the State of the Brain*, *supra*.

response to nociceptive, or painful, stimuli, are used as the principal markers of potentially inadequate general anesthesia.[69]

66. In other words, if the brain stem, is adequately shut down, as measured by a lack of change in heart rate or blood pressure, an individual is generally considered adequately anesthetized such that they will likely not experience pain or suffering in response to noxious stimuli. In order to avoid pain, anesthesiologists therefore aim to put the brain stem "to sleep", in order to ensure that the patient, although non-communicative, is nonetheless not experiencing pain or suffering.[70]

67. "Other signs of inadequate antinociception" or failure to block the pain pathway, "are perspiration, tearing, pupil dilation, and return of muscle tone and movement. However, changes in muscle tone and movement are not observed" where an individual is receiving a muscle relaxant or a paralytic agent. [71]

68. The professional standard is thus that it is not sufficient to relieve only the "affective" or subjective component of pain but rather to also eliminate any perception of pain, or nociception. In order to achieve this standard, the goal of the anesthesiologist is to use a drug protocol that will put the brain stem to sleep, not only the cerebrum. If the cerebrum is sufficiently blocked, but pain reception is not, an individual exposed to a sufficiently painful stimulus may not experience suffering, but will experience pain.

69. Thus, the goal of general anesthesia is to create a condition marked by antinociception, unconsciousness, amnesia, and immobility, even in the presence of repeated

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[69] Brown, et al., *Monitoring the State of the Brain*, *supra*;

[70] Brown, et al., *Monitoring the State of the Brain*, *supra.*

[71] Brown, et al., *Monitoring the State of the Brain*, *supra;* Parsons, *supra*.

painful stimuli. This state requires the individual to have attained a state of sedation which is deeper than when an individual is deemed unresponsive to verbal or tactile stimulus.

3. Anesthetic Induction, Maintenance, and Emergence

70. Anesthesia typically involves three stages: Induction, Maintenance, and Emergence.

71. "The induction of anaesthesia refers to the transition from an awake to an anaesthetized state. This end point can be ill defined and the process of induction is a time of physiological disruption with multi-system effects."[72] Physiological signs of induction of general anesthesia include loss of the ability of smooth pursuit with the eyes when asked to track a finger; and loss of the oculocephalic reflex and corneal reflex.[73] "The oculocephalic reflex is assessed by turning the patient's head from side to side, while lifting the eyelids. Before administration of the induction anesthetic, when the reflex is intact in a patient with no neurologic deficits, the eyes move in the direction opposite the motion of the head. When the reflex is lost, the eyes stay fixed in the midline."[74] "The corneal reflex has traditionally been assessed using a wisp of cotton at the corner of the eye to stroke the cornea. An easier way to assess the reflex is to allow a drop of sterile water to fall on the cornea. . . . With either approach, the reflex is intact if the eyes blink consensually, is impaired if there is a blink in one eye and not the other, and is absent if there is no blink."[75]

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[72] Donohue, et al., *An introduction to anaesthesia*, British Journal of Hospital Medicine, May 2013, Vol. 74, No. 5, C71-C75.

[73] Brown, et al., *Monitoring*, *supra*.

[74] Brown, et al., *Monitoring*, *supra*.

[75] Brown, et al., *Monitoring*, *supra*.

72. Maintenance of anaesthesia refers to keeping a patient in an anesthetic and insensate state. Maintenance "can be achieved using inhaled volatile agents or continuous infusion of intravenous agents."[76] "[T]he physiologic signs of changes in heart rate, arterial blood pressure, and movement are the measurements most commonly used to track the anesthetic state during maintenance of general anesthesia. When the state of general anesthesia is not adequate for the level of surgical (nociceptive) stimulation, heart rate and arterial blood pressure can increase dramatically. The changes in heart rate and arterial blood pressure that anesthetized patients show in response to a nociceptive stimulus can be explained in terms of the" nociceptive or pain pathways discussed above.[77]

73. "Once anaesthesia is no longer required, maintenance agents can be switched off. Before emergence, adequate analgesia and anti-emesis [anti-nauseals] should be ensured and neuromuscular junction function restored if a muscle relaxant has been used. Like induction, emergence can be a time of physiological disturbance. As patients start to wake from anaesthesia or 'lighten' they may develop agitation, laryngospasm and breath-holding."[78] "The state of the patient during emergence from general anesthesia can be tracked reliably by monitoring the patient's physiologic signs and performing neurologic examinations. Many of these physiologic changes relate to the return of brainstem function."[79] Other signs that a subject is emerging from general anesthesia include "swallowing, gagging, salivation, tearing, and grimacing."[80] "Opening of the eyes is typically one of the last physiologic signs observed in patients emerging from general

[76] Donohue, et al., *supra*.

[77] Brown, et al., *Monitoring*, *supra*.

[78] Donohue, et al., *supra*.

[79] Brown, et al., *Monitoring*, *supra*.

[80] Brown, et al., *Monitoring*, *supra*.

anesthesia. In particular, patients may respond reliably to verbal commands, have substantial return of motor functions, yet not necessarily open their eyes. In general, patients tend to keep their eyes closed even when consciousness has returned."[81]

4. General Anesthesia and Levels of Sedation

74. As outlined by the American Society of Anesthesiologists, medications used to produce changes in consciousness ranging from anxiolysis (anxiety relief) to general anesthesia can be considered on a continuum, illustrated by the chart below:

Continuum of Depth of Sedation:
Definition of General Anesthesia and Levels of Sedation/Analgesia*

Committee of Origin: Quality Management and Departmental Administration

(Approved by the ASA House of Delegates on October 13, 1999, and last amended on October 23, 2019)

	Minimal Sedation Anxiolysis	*Moderate Sedation/ Analgesia ("Conscious Sedation")*	*Deep Sedation/ Analgesia*	*General Anesthesia*
Responsiveness	Normal response to verbal stimulation	Purposeful** response to verbal or tactile stimulation	Purposeful** response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

[81] Brown, et al., *Monitoring*, *supra*.

75. The following definitions are important to understanding the depths of anesthesia:

a. **Minimal Sedation (Anxiolysis)** is a drug-induced state during which patients respond normally to verbal commands. Although cognitive function and physical coordination may be impaired, airway reflexes, and ventilatory and cardiovascular functions are unaffected.[82]

b. **Moderate Sedation/Analgesia** ("Conscious Sedation") is a drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.[83]

c. **Deep Sedation/Analgesia** is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.[84]

d. **General Anesthesia / Anesthetic**: General Anesthesia is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often

[82] American Society of Anesthesiologists, Committee on Quality Management and Departmental Administration Continuum-of-depth-of-sedation-definition, October 23, 2019. Available here.

[83] American Society of Anesthesiologists, Committee on Quality Management and Departmental Administration Continuum-of-depth-of-sedation-definition, October 23, 2019. Available at here.

[84] American Society of Anesthesiologists, Committee on Quality Management and Departmental Administration, *Continuum-of-depth-of-sedation-definition*, October 23, 2019. Available at here.

require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.[85]

76. With minimal sedation, or anxiolysis, the patient will have a normal response to verbal stimulation without effect on airway, ventilation or cardiovascular function.[86] This level of sedation is often used for procedures such as MRI, to help with claustrophobia; sometimes for injections, for anxiety; or for those who have anxiety over the dentist. Minimal sedation does not usually require a separate individual to monitor the subject receiving sedation.

77. With moderate sedation, often termed conscious sedation, the individual will maintain purposeful response to verbal or tactile stimulation. This is the first level of sedation that requires an individual to monitor the subject receiving sedation separate from the physician performing the procedure. The airway is maintained without medical intervention with adequate respiration and stable hemodynamics.[87] This level of sedation is often used for procedures with low pain stimulus, such as colonoscopy or dental procedures.

78. Deep sedation is described as a state at which the individual will demonstrate purposeful response to repetitive or painful stimulation. In this state intervention may be required to maintain the airway, and ventilation may be impaired.[88] This level of sedation is might be used during surgery done under local anesthetic or regional block.

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[85] American Society of Anesthesiologists, Committee on Quality Management and Departmental Administration, *Continuum-of-depth-of-sedation-definition*, October 23, 2019. Available at here.

[86] American Society of Anesthesiologists, Quality Management and Departmental Administration, *Continuum of Depth of Sedation: Definition of General Anesthesia and Levels of Sedation/Analgesia*, last amended Oct. 23, 2019.

[87] American Society of Anesthesiologists, *Continuum of Depth of Sedation*.

[88] American Society of Anesthesiologists, *Continuum of Depth of Sedation*.

79. General anesthesia is obtained only when the individual is unarousable to painful stimulus. The definition of general anesthesia "is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation."[89] During general anesthesia, a subject's "ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired."[90] This level of sedation is often used for abdominal, heart, or thoracic surgery.

80. The noxious stimuli of the Execution Protocol is at least equal to that of surgical stimulus. The same level of depth of anesthesia required for surgery—general anesthesia—is therefore necessary to avoid a substantial risk of severe pain and suffering.

5. Determination of Depth of Anesthesia

81. Depth of anesthesia can be monitored by a variety of maneuvers and mechanisms. Verbal stimulation may be the first step in assessing depth of sedation, and patients in the first two categories of sedation above, minimal or moderate sedation, would likely respond.

82. In an individual, tactile stimulation may be invoked. Often the eyelash reflex is used as a method of tactile stimulation. This involves gently brushing or stroking the eyelash and looking for a blink response. With deeper levels of sedation, stimulation such as sternal rub or pressure on the nail bed may be used to invoke a noxious, or nociceptive, stimuli.

[89] American Society of Anesthesiologists, *Continuum of Depth of Sedation*.

[90] American Society of Anesthesiologists, *Continuum of Depth of Sedation*.

83. Minimum alveolar concentration, or MAC, is a measure used to judge depth of anesthesia.[91] When inhalation anesthetics are used to induce or maintain general anesthesia the concentration of anesthetic necessary to attain general anesthesia is quantified as the minimum alveolar concentration, or MAC. MAC is the minimum alveolar concentration at which 50% of individuals will not move in response to a surgical stimulus. It has been demonstrated, however, that when end-tidal anesthetic agent [the concentration exhaled by a subject] is approximately 0.33 MAC, 50% of subjects do not respond appropriately to oral commands.[92] This study demonstrates that at a dose that is only one-third of the dose that would put 50% of patients in a state of general anesthesia, 50% of patients are nonetheless unresponsive to oral commands. The significance of this is that a patient may be unresponsive to verbal stimulus but nevertheless be able to experience pain.

84. As outlined by Brown, et al., various physiologic signs can also be used to monitor the depth of anesthesia and loss of consciousness. At the initiation of loss of consciousness, or LOC, the subject may lose lateral excursions of the eyes when asked to track a finger, termed loss of smooth pursuit.[93] Nystagmus, or uncontrolled movements of the eyes, may appear. Blinking may increase and eyes may fix in midline.

85. The corneal and oculocephalic reflexes are also used to assess depth of sedation and anesthesia. Corneal reflex is a reaction of the eye when the cornea is stimulated. The corneal reflex is intact when the eyes blink consensually or together; is considered impaired when only when only one eye blinks; and is absent with no blink. It is often assessed by stroking the cornea

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[91] Melvin MA, et al. *Induction of anesthesia with midazolam decreases halothane MAC in humans*. Anesthesiology 1982; 57:238-41.

[92] Avidan, et al., *supra.*

[93] Brown, et al., *General Anesthesia, Sleep, and Coma*, N Engl J Med 2010;363:2638-50.

with a wisp of cotton or a drop of water.[94] The oculocephalic reflex can also be monitored. In the intact state with the eyes open the eyes move in the opposite direction when the head is turned side to side. When impaired the eyes stayed fixed in midline.

86. Both the corneal and oculocephalic reflexes are lost with the induction and maintenance of general anesthesia due to the effect of anesthetic agents on the brainstem and the loss of suppression of the corresponding reflexes. The loss of these reflexes occurs with the loss of responsiveness, and may be consistent with a loss of consciousness.

87. Apnea, or cessation of respiration, and loss of muscle tone, are other measures of depth of anesthesia. As with the corneal and oculocephalic reflexes, these physiologic changes also correspond to action of drugs at sites in the brainstem and spinal cord.[95]

88. Heart rate and blood pressure are also monitored during surgery and used as a measure of the adequacy of depth of anesthesia. "Movement and the physiological responses of changes in heart rate, blood pressure, and perhaps respiratory rate, are the most commonly used markers of nociception."[96] When anesthesia depth is inadequate, the nociceptive stimulus can lead to a marked increase in heart rate and blood pressure – each a sign the patient experiences pain and suffering. It is believed that this is due to nociceptive system, as explained above. Other signs of inadequate anesthesia, and often seen on emergence from general anesthesia, include tearing or lacrimation, sweating, salivation, gagging, and vocalization or moaning and grimacing, which are also signs of the patient experiencing pain and/or suffering. All of these may occur with return of

[94] Brown, et al., *Monitoring the State of the Brain and Central Nervous System During General Anesthesia and Sedation, in Miller's Anesthesia*, 9th Edition (Gropper, ed.).

[95] Brown, et al., *Monitoring the State of the Brain*.

[96] Brown, et al., *Monitoring the State of the Brain*.

brainstem functions.[97]

VI. PRISONERS EXECUTED IN ACCORDANCE WITH OKLAHOMA'S EXECUTION PROTOCOL, FACE A SUBSTANTIAL RISK OF EXPERIENCING SEVERE PAIN AND SUFFERING.

A. Midazolam Cannot Reliably Induce or Maintain General Anesthesia in the Presence of Painful and Noxious Stimuli, Including as Caused by the Second and Third Drugs in Oklahoma's Three-Drug Lethal Injection Protocol.

1. Midazolam Cannot Be Used Alone to Reliably Induce General Anesthesia.

89. As explained above, Section V.A.1, midazolam is not routinely used as the only drug to induce general anesthesia in order to render a subject insensate and in an anesthetic state because it is unreliable for use as a sole induction agent.[98] If midazolam is used as an induction agent, it is almost always used in combination with other drugs for inducing general anesthesia and rendering a patient insensate,[99] with the exception of procedures involving "conscious sedation," such as colonoscopy or dental procedures.[100]

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[97] Brown, et al., *Monitoring the State of the Brain*.

[98] Gamble, et al., *Evaluation of midazolam as an intravenous induction agent in dogs, Anaesthesia*, 1981, Volume 36, pages 868-873 (midazolam administered with dexmedetomidine or other medications); Khanderia U, Pandit SK(1987) *Use of midazolam hydrochloride in anesthesia.* Clin Pharm. 6:533-547; Samuelson, et al., *Hemodynamic Responses to Anesthetic Induction with Midazolam or Diazepam in Patients with Ischemic Heart Disease*, Anesthesia and Analgesia, Vol. 80, No. 11, November 1981; 60:802-9; White, *Comparative Evaluation of Intravenous Agents for Rapid Sequence Induction – Thiopental, Ketamine, and Midazolam*, Anesthesiology 57:279-284, 1982.

[99] *E.g.*, Giovannitti JA, Trapp LD (1991) *Adult sedation: oral, rectal, IM, IV*. Anesth Prog. 38:154-71 (clinical effects are highly variable); Khanderia U, Pandit SK(1987) *Use of midazolam hydrochloride in anesthesia.* Clin Pharm. 6:533-547 (opiate pretreatment makes induction more consistent); *see also* Samuelson, et al., *Hemodynamic Responses to Anesthetic Induction with Midazolam or Diazepam in Patients with Ischemic Heart Disease*, Anesthesia and Analgesia, Vol. 80, No. 11, November 1981; 60:802-9.

[100] *E.g.*, Hoffman MS, Butler TW, Shaver T. *Colonoscopy without sedation*. J Clin Gastroenterol 1998; 26:279-82; Giovannitti JA, Trapp LD (1991) *Adult sedation: oral, rectal, IM, IV*. Anesth Prog. 38:154-71 (discussing conscious sedation in dental procedures); Reves, et al., *Midazolam: Pharmacology and Uses*, Anesthesiology Vol. 62, No. 3 Mar. 1985, 62:310-324;

90. Published literature repeatedly confirms that midazolam is unreliable as a sole induction agent:[101]

- "Induction of anesthesia with midazolam alone is somewhat unpredictable; opiate pretreatment makes induction more consistent."[102]
- "Although benzodiazepines used for IV sedation have a high margin of safety, their clinical effects are highly variable."[103]
- "After the administration of fentanyl or fentanyl plus dehydrobenzperidol anaesthesia could be induced easily [with midazolam]."[104]

91. Research on other benzodiazepines also confirms that as a class, these drugs, which include midazolam, are unreliable at best for induction of general anesthesia. For example, due to its variability of onset, unreliable duration of sedative action, lack of reliable production of general anesthesia rendering an individual insensate, and suppression of awareness during surgery, among other things, valium is no longer commonly used in most modern anesthesia practices.[105] Because the drugs in the class benzodiazepines are consistent in their mechanism of action, and qualitatively similar in their effects, studies involving one benzodiazepine can reflect similar behavior among the class of benzodiazepines.[106]

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[101] *See also* Bai, et al., *Distinct Functional and Pharmacological Properties of Tonic and Quantal Inhibitory Postsynaptic Currents Mediated by γ-Aminobutyric AcidA Receptors in Hippocampal Neurons*, Molecular Pharmacology Vol. 59, No. 4, 814-824, 2001.

[102] Khanderia U, Pandit SK(1987) *Use of midazolam hydrochloride in anesthesia.* Clin Pharm. 6:533-547.

[103] Giovannitti JA, Trapp LD (1991) *Adult sedation: oral, rectal, IM, IV*. Anesth Prog. 38:154-71; Khanderia U, Pandit SK(1987) *Use of midazolam hydrochloride in anesthesia.* Clin Pharm. 6:533-547.

[104] Kanto J, Sjovall S, Vuori A. *Effect of different kinds of premedication on the induction properties of midazolam*. Br J Anaesth 1982; 54:507-11.

[105] Ochs HR, Greenblatt DJ, Lauven PM et al. *Kinetics of high-dose i.v. valium*. Br J Anaesth 1982; 54:849-52.

[106] *See, e.g.*, Mihic, *supra*.

92. Because of its unreliability and unpredictability with respect to induction of anesthesia, midazolam is rarely used as a sole induction agent for general anesthesia.

2. Midazolam Is Not Approved for or Used to Maintain General Anesthesia After Induction.

93. Additionally, midazolam is neither used nor approved for maintenance of general anesthesia as a sole agent.[107] Therefore, even if anesthesia is successfully induced using only midazolam, a subject will not remain in an insensate and anesthetic state after they are subjected to even mild pain stimuli.

94. The FDA-approved indications for use affirm that midazolam is not approved for maintenance of general anesthesia at any dose. Midazolam is approved only for (1) "preoperative sedation/anxiolysis/amnesia"; (2) "sedation anxiolysis/amnesia prior to or during diagnostic, therapeutic or endoscopic procedures," such as those requiring only "conscious sedation"; (3) "induction of anesthesia, before administration of other anesthetic agents"; or (4) "sedation of intubated and mechanically ventilated patients as a component of anesthesia or during treatment in a critical care setting."[108]

95. Numerous studies confirm that midazolam is not appropriate for use alone for maintenance of general anesthesia. For example:

- "The relatively short elimination half-life (th) of approximately ***2*** hours and the brief hypnotic effect of intravenous midazolam (with some patients beginning to arouse **5** to **10** minutes after receiving intravenous injections) prompted our

[107] Samuelson, et al., *Hemodynamic Responses to Anesthetic Induction with Midazolam or Diazepam in Patients with Ischemic Heart Disease, Anesthesia and Analgesia*, Vol. 80, No. 11, November 1981; 60:802-9 ("[M]idazolam and diazepam induction require supplementation with narcotics or inhalation anesthesics for maintenance of anesthesia.")

[108] Midazolam Hydrochloride—midazolam hydrochloride injection, solution, Hospira, Inc. (official label), available at https://dailymed.nlm.nih.gov/dailymed/fda/fdaDrugXsl.cfm?setid=b95415fa-17c2-42ab-a6b0-e628d01c94ed&type=display.

combination of N_20 with midazolam. . . . [M]idazolam and diazepam induction require supplementation with narcotics or inhalation anesthesics for maintenance of anesthesia. Our data indicate that 50% nitrous oxide and oxygen plus midazolam is a hemodynamically stable combination during anesthetic induction, but is not sufficient to block totally the hemodynamic effects of orotracheal intubation."[109]

- "In higher doses anaesthesia can be induced and maintained *under comedication with an analgesic narcotic*."[110]
- "Both propofol and midazolam obtund memory and consciousness but only propofol produces a level of neurodepression sufficient to prevent movement in response to painful stimuli."[111]
- "We found midazolam to be a mild hypnotic which requires powerful premedication when used alone as an anaesthetic agent for minor surgery.[112]

96. Drug overdose studies further confirm that some subjects that take even very large doses of benzodiazepines are still responsive to even mild pain stimulus.[113] This is consistent with

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[109] Samuelson, et al., *Hemodynamic Responses to Anesthetic Induction with Midazolam or Diazepam in Patients with Ischemic Heart Disease, Anesthesia and Analgesia*, Vol. 80, No. 11, November 1981; 60:802-9.

[110] Amrein & Hetzel, *supra*.

[111] Bai, et al., *Distinct Functional and Pharmacological Properties of Tonic and Quantal Inhibitory Postsynaptic Currents Mediated by γ-Aminobutyric AcidA Receptors in Hippocampal Neurons*, Molecular Pharmacology Vol. 59, No. 4, 814-824, 2001.

[112] Kanto J, Sjovall S, Vuori A. *Effect of different kinds of premedication on the induction properties of midazolam*. Br J Anaesth 1982; 54:507-11.

[113] Divoll M et al. (1981) *Benzodiazepine overdosage: plasma concentrations and clinical outcome*. Psychopharmacology (Berl) 73:381-383 (twelve patients who overdosed on a benzodiazepine alone (diazepam), in doses as high as 750 mg, suffered no ill effects, were minimally sedated, and discharged from the emergency department within 24 hours); Allen MD et al. (1980) *Pharmacokinetic study of lorazepam overdosage*. Am J Psychiatry 137:1414-1415 (patients who overdosed on another benzodiazepine, lorazepam, were admitted to the ER with blood levels 12 times greater than therapeutic concentrations; all responded to a light pain stimulus); Greenblatt DJ et al. (1977) *Acute overdosage with benzodiazepine derivatives.* Clin Pharmacol Ther. 21497-514 (of twelve benzodiazepine overdose patients with no other drugs in their system, none needed assisted ventilation and all were discharged within two days with no ill effects; these included doses of 2.5 grams of chlordiazepoxide (Librium®) and 400 mg of diazepam (Valium®)).

the mechanism of action and relatively high safety profile of the benzodiazepines, including midazolam.

97. Awareness episodes during anesthetics involving midazolam with paralytic agents have been shown to occur in over 71% of patients in one study, despite that the anesthesiologists believed that their patients were unconscious and unaware at the time.[114] In another study, more than 40% of patients who had awareness indicated they also experienced pain.[115]

3. Midazolam Does Not Provide Pain Relief and May Enhance Pain.

98. Midazolam alone is also incapable of rendering a subject insensate to pain, and may, in some circumstances, actually enhance pain. Benzodiazepines, including midazolam, are not analgesic, meaning that they do not reduce pain. In fact, benzodiazepines can be an anti-analgesic, causing an increase in the perception of pain.[116] Benzodiazepines can block the effectiveness of opioids, which suggests they likely also block the effectiveness of a subject's natural pain relief mechanisms.[117] Other paradoxical reactions to benzodiazepines are also common and include delirium, combativeness, paradoxical fear, anxiety and panic.[118]

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[114] Russell IF. *The ability of bispectral index to detect intra-operative wakefulness during isoflurane/air anaesthesia, compared with the isolated forearm technique. Anaesthesia. 2013;68:1010-1020.*

[115] Russell IF. Comparison of Wakefulness with Two Anaesthetic Regimens: Total I.V. v. Balanced Anaesthesia. British Journal Anaesth. 1986;58:965-968.

[116] Brown, et al., *General Anesthesia, Sleep, and Coma*, N Engl J Med 2010;363:2638-50; Gear, et al., *Benzodiazepine mediated antagonism of opioid analgesia*, Pain 71 (1997) 25–29; Wright, *Limited Utility for Benzodiazepines in Chronic Pain Management: A Narrative Review*, Adv Ther (2020) 37:2604–2619.

[117] *Id.* [ADDITIONAL SUPPORT].

[118] Brown, et al., *General Anesthesia, Sleep, and Coma*, N Engl J Med 2010;363:2638-50.

99. Therefore, midazolam will be ineffective for mitigating any painful stimuli, including the pain and suffering caused by the third drug in Oklahoma's three-drug lethal injection protocol.

4. Midazolam Has a Recognized Ceiling Effect Below the Dose Required by Oklahoma's Lethal Injection Protocol.

100. In addition, midazolam has been described as having an observed "ceiling effect" such that after a certain dose, any increase in the amount or concentration administered will have little to no additional effect.[119] For example, in non-clinical settings, researchers have noted:

- "The maximum obtainable enhancement of GABAergic transmission with benzodiazepines is limited and occurs at about 10 times the threshold doses [0.1 to 0.8 mg/kg for midazolam]."[120]
- "Examination of the concentration-response relationship . . . for the enhancement of the tonic current by midazolam indicated that concentrations of greater than 0.2 mM caused no further increase in current amplitude. . . . Unlike midazolam, the response to propofol did not saturate but continued to increase with concentrations over the range tested (0.2–5 mM), as described previously (Orser et al., 1994)."[121]
- "A ceiling effect is observed after maximal doses of midazolam"[122]

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[119] *See, e.g.*, Bai, et al., *Distinct Functional and Pharmacological Properties of Tonic and Quantal Inhibitory Postsynaptic Currents Mediated by γ-Aminobutyric AcidA Receptors in Hippocampal Neurons*, Molecular Pharmacology Vol. 59, No. 4, 814-824, 2001; *id.* ("Unlike midazolam, the response to propofol did not saturate but continued to increase with concentrations over the range tested); Coldwell, et al., *Acute Tolerance and Reversal of the Motor Control Effects of Midazolam*, Pharmacology Biochemistry and Behavior Vol. 59, no. 2, pp. 537-545, 1998; Ibrahim AE et al. (2002) *The influence of parecoxib, a parenteral cyclooxygenase-2 specific inhibitor, on the pharmacokinetics and clinical effects of midazolam*. Anesth Analg. 95:667-673; Kuizenga K et al. (2001) *Biphasic EEG changes in relation to loss of consciousness during induction with thiopental, propofol, etomidate, midazolam or sevoflurane*. Br J Anaesth. 86:354-360; [TAB 33] Miyake W et al. (2010) *Electroencephalographic response following midazolam-induced general anesthesia: relationship to plasma and effect-site midazolam concentrations*. J. Anesthesia 24:386-393; *see also* Amrein & Hetzel, *supra*.

[120] Gamble, *supra*.

[121] Bai, et al., *supra*.

[122] Amrein & Hetzel, *supra*.

- "Benzodiazepines do not work directly on the GABA receptor, so there is a physiologic ceiling effect, which contributes to their safety and low toxicity"[123]

- "The apnoeic threshold was increased by the lowest midazolam dose but not any further when additional doses of midazolam were administered, i.e., demonstrating a ceiling effect."[124]

- "Benzodiazepines have no direct effect but require GABA to be present. If $GABA_A$ receptors are fully saturated by GABA during long term sedation, benzodiazepines will no longer achieve deeper sedation resulting in a so-called ceiling effect"[125]

101. Defendants in this action have also recognized that midazolam has a potential ceiling effect.[126] ODOC Director Crow testified that the ceiling effect is "the points at which a drug reaches its maximum effectiveness, no matter the dose, from that point forward."[127] Director Crow testified:

> Although I can't recall specifics, there's opinions that vary from the amount being something in the neighborhood of 40 to 50 milligrams and higher. There are others that I had seen that have stated that the ceiling effect occurs at a lower dosage, but typically the number that I have seen is 40 to 50 milligrams.[128]

Director Crow further testified: "Those are the dosages that I have reviewed, but that does not eliminate the possibility that there are other opinions higher than that."[129]

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[123] Majumdar JR et al. *Effects of midazolam on postoperative nausea and vomiting and discharge times in outpatients undergoing cancer-related surgery*. AANA Journal (2019) 87(3):179-183.

[124] Schwieger JM et al. *Intrathecal midazolam reduces isoflurane MAC and increases the apnoeic threshold in rats*. Can J Anaeth. (1994) 41:144-188.

[125] Michel J et al., *Gamma-hydroxybutyrate: Is it a feasible alternative to midazolam in long-term mechanically ventilated children?* Curr Med Res Opin. (2019) 35:1721–1726.

[126] 11/12/2020 Dep. Tr. 182:18-21, 183:7-12.

[127] *Id.* 183:10-11.

[128] *Id.* 184:2-7.

[129] *Id.* 184:4-6.

102. This observed "ceiling effect" is consistent with midazolam's known mechanism of action. As explained above, Section V.A.1, midazolam, like all benzodiazepines, requires the presence of GABA to have an effect on the $GABA_A$ receptors. Midazolam is therefore dependent on the availability and concentration of both GABA and the $GABA_A$ receptors themselves. In contrast, barbiturates do not require the presence of GABA to have an effect. The phenomenon of the "ceiling effect" is well recognized for the benzodiazepines generally,[130] and midazolam in particular.

5. Individual Response to Midazolam Doses Varies Significantly.

103. In addition, the individual variation in response to the same dose of midazolam is well documented,[131] independent from the observed ceiling effect.

- [Two patients] initially both displayed poor responses to midazolam at doses that would ordinarily have been effective. With significant dose titration, there were some periods of adequate response. However, these periods were short-lived and rapid dose escalation, as well as the addition of other sedating medications, was required. The poor responses in these individuals are consistent with several reports indicating considerable inter-individual variation in response to midazolam and other sedatives."[132]

- "The pharmacokinetics of midazolam are affected by obesity, age, heptaic cirrhosis, and severity of critical illness."[133]

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[130] For example, like midazolam, the pharmacokinetics (how the drug is taken up, metabolized and cleared from the body) of intravenous diazepam are unaffected by dose, even at very high doses. Ochs HR, Greenblatt DJ, Lauven PM et al. *Kinetics of high-dose i.v. valium*. Br J Anaesth 1982; 54:849-52.

[131] E.g., Gamble, et al., *Evaluation of midazolam as an intravenous induction agent*, Anaesthesia, 1981, Volume 36, pages 868-873, 872; Cheng, et al., *When Midazolam Fails, Journal of Pain and Symptom Management*, Vol. 23 No. 3 March 2002, at Table 2; Kanto J, Sjovall S, Vuori A. *Effect of different kinds of premedication on the induction properties of midazolam*. Br J Anaesth 1982; 54:507-11.

[132] Cheng, et al., *When Midazolam Fails, Journal of Pain and Symptom Management*, Vol. 23 No. 3 March 2002, at 261.

[133] Vuyk, et al., *supra*.

104. Some "factors responsible for the diversity in response may include concomitant medications, age, concurrent disease, overall health status, alcohol use, liver disease, renal disease, smoking and hormonal status."[134]

105. One contributing factor to the variation in individual response is individual genetic variation, which results in differing receptor subtypes present among individuals.[135] The diagram below shows an analysis of the 19 known genes that can code for the human $GABA_A$ subunits: [136]

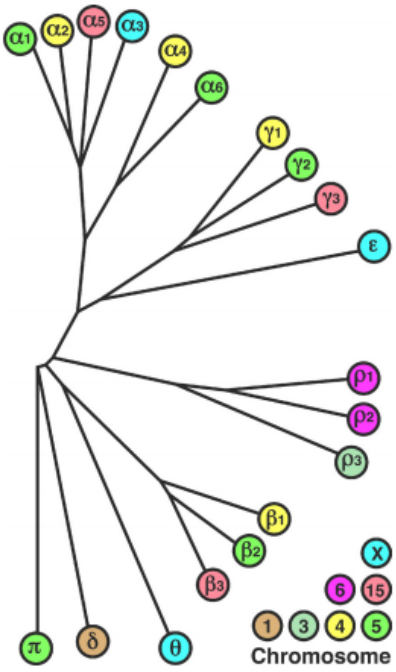

FIGURE 1. **Phylogenetic tree analysis of the 19 known genes coding for human $GABA_A$ receptor subunits.** The immature amino acid sequences were obtained from the UniProt database (94). The alignment was done with ClustalX (95), and Dendroscope (64) was used for depiction of the dendrogram.

[134] Cheng, et al., *When Midazolam Fails, Journal of Pain and Symptom Management*, Vol. 23 No. 3 March 2002, at 261.

[135] Cheng, et al., *When Midazolam Fails, Journal of Pain and Symptom Management*, Vol. 23 No. 3 March 2002; Ticku, *Benzodiazepine-GABA Receptor-Ionophore Complex: Current Concepts*, Neuropharmacology Vol. 22, No. 12, 1459-1470.

[136] Sigel and Steinmann, JBC, Papers in Press, Oct. 4, 2012, J. Biol. Chem. Vol. 287, No. 48, pp. 40224-40231.Structure, Function, and Modulation of $GABA_A$ Receptors.

106. The effects of a single dose of midazolam may vary significantly between individuals due to this genetic variation:

> Considerable heterogeneity exists among human $GABA_A$ receptors; this heterogeneity is thought to contribute to the myriad effects of these agents in vivo. Because receptor subunit composition appears to govern the interaction of various allosteric modulators with these channels, there has been a surge in efforts to find agents displaying different combinations of benzodiazepine-like properties that may reflect selective actions on one or more subtypes of $GABA_A$ receptors. A number of distinct mechanisms of action, reflecting involvement of specific subunits of the $GABA_A$ receptor, likely contribute to distinct effects of various benzodiazepines—the sedative-hypnotic, muscle-relaxant, anxiolytic, amnesic, and anticonvulsant effects.[137]

107. As shown in **Figure 1**, in section V.A.1, the $GABA_A$ receptor is a transmembrane protein, meaning it spans the width of a cell membrane, and is made up of five subunits. Each of the five subunits can in turn differ, making the number of possible variations among individuals' receptor types quite large, at least in the dozens.[138]

[137] Mihic, supra; Bowery, et al., *GABA Receptor Multiplicity: Visualization of Different Receptor Types in the Mammalian CNS*, Neuropharmacology Vol. 23, No. 2B, pp. 219-231, 1984; *see also* Cheng, *supra*.

[138] Mihic, *supra*; Bowery, et al., *supra*.

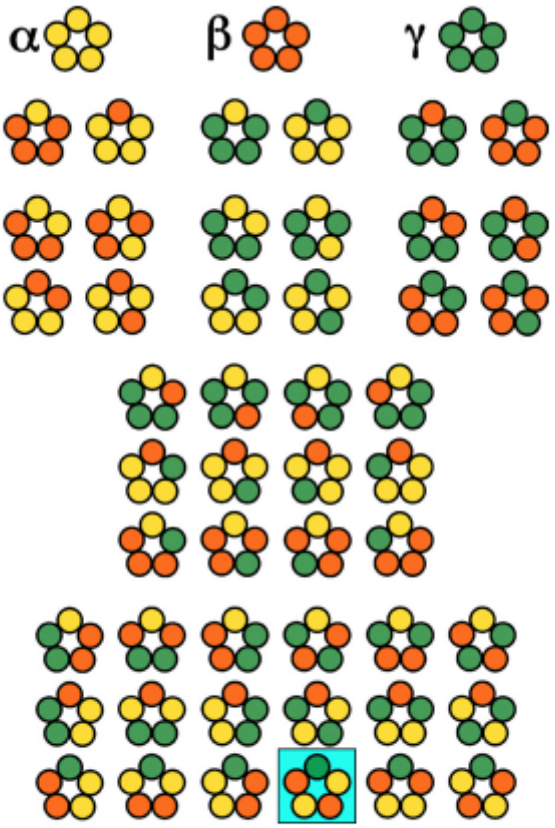

FIGURE 3. **Possible arrangements in a pentamer of subunits taken from three different types, α (*yellow*), β (*red*), and γ (*green*).** There are three homomeric receptors, 18 receptors composed of two subunits, and 30 receptors composed of three different subunits. The receptor on the *blue square* represents the current consensus of the subunit arrangement in $\alpha_1\beta_2\gamma_2$ $GABA_A$ receptors as seen from the cell exterior.

108. Another factor contributing to the variation in response between individuals may be interaction with other drugs, specifically those causing induction of the CYP 3A4 isoenzyme. Midazolam "undergoes biotransformation by the P450 system," which is conducted primarily in the liver.[139] The presence of other drugs that stimulate, or induce, the P450 system may cause benzodiazepines, including midazolam, to be metabolized, or broken down more rapidly.[140] This results in less of the effective form of the benzodiazepine being available. Drugs that can induce the P450 system include barbiturates, glucocorticoids, phenobarbital, rifampin, commonly used to

[139] Cheng, *supra*.

[140] E.g., Vuyk, et al., *supra*, at 657.

treat tuberculosis, or pioglitazone and troglitazone, each used to treat diabetes. Thus, in addition to the individual variation noted elsewhere in this section, drug interaction can cause midazolam to be removed from an individual's system more rapidly than expected, leading to diminished effects.

109. Additional external and internal factors can affect individual response to the same therapeutic dose of benzodiazepines, including midazolam. Such factors include

> the intensity and nature of external stimuli, the uptake and neuronal processing of a stimulus, and the amount of GABA available and necessary to cope with the stimulus. Although diagnostic and surgical procedures are to a great extent standardised within each hospital unit, and when dealing with a patient attempts are made to protect him from exciting stimuli, each patient's perception of what happens around him, or may happen to him is different. As a result, patients may react differently to equal doses of a given BZD or ***vice versa*** different doses may be needed to achieve the same effect in different patients.[141]

110. Other studies also suggest that subjects may develop acute or sudden tolerance to midazolam when given a bolus and constant infusion administration. Subjects tested for performance showed improvement on initially impaired performance despite receiving a gradual increase in midazolam concentrations.[142] And as with other benzodiazepines, prior or concomitant prescription or illicit drug use by the recipient can lead to cross-tolerance (requiring markedly higher doses to produce its usual effect), or to synergistic or additive actions.

111. To summarize, midazolam (1) cannot reliably be used alone to induce general anesthesia; (2) is neither approved nor used to maintain general anesthesia; (3) has no analgesic, or pain relieving properties; (4) is associated with an observed "ceiling effect," which is consistent

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[141] Amrein & Hetzel, *Pharmacology of Dormicum® (midazolam) and Anexate® (flumazenil)*, Acta Anasesthesiol Scand 1990: 34, Supplementum 92:6-15.

[142] Coldwell, et al., *supra*.

with the mechanism of action of midazolam and other benzodiazepines, that suggests midazolam's effects do not increase above a certain dose that is far below that called for in Oklahoma's execution protocol; and (5) demonstrates wide dose response variability requiring individualized dosing. Midazolam is therefore not sufficient alone to maintain a patient in an insensate and anesthetic state in the presence of painful or noxious stimuli,[143] such as the pain and suffering caused by the second and third drugs in Oklahoma's three-drug execution protocol.

B. Midazolam Cannot Reliably Render and Maintain a Prisoner in an Insensate and General Anesthetic State During Flash Pulmonary Edema.

112. For all of the reasons explained directly above, midazolam is not sufficient, when used alone, to maintain a patient in an insensate state and in general anesthesia in the presence of painful or noxious stimuli. Pulmonary edema, which is found in the autopsies of almost all prisoners executed using midazolam, and specifically using the three-drug protocol Oklahoma plans to use, is such a noxious stimulus.

1. Acute or "Flash" Pulmonary Edema Causes Severe Suffering.

113. Pulmonary edema (or fluid in the lungs) is a process in which fluid from the bloodstream floods into the lungs, causing failure of the lungs to transfer sufficient oxygen into the bloodstream.[144] In addition, the lungs "stiffen" because of the increased fluid, and it becomes physically harder and harder for the victim to draw breath. Actual spasm of the airways (bronchospasm) occurs as the swelling ("edema") of airway passages makes breathing increasingly

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[143] Reves, et al., *Midazolam: Pharmacology and Uses*, Anesthesiology Vol. 62, No. 3 Mar. 1985, 62:310-324;

[144] Wedro B. Pulmonary edema. Emedicinehealth. Updated 2/1/19. Available at: http://emedicinehealth.com/pulmonary_edema/article_em.htm#what_are_the_symptoms_of_pulmonary_edema . Accessed January 1, 2021; Gagne D. *Acute pulmonary edema*. CathLabDigest September 5, 2012. Volume 20. Available at: https://cathlabdigest.com/Acute-Pulmonary-Edema. Accessed Dec. 28, 2020.

difficult, and as the airways themselves swell shut from the fluid. The victim is literally drowning, and if aware will suffer sensations of shortness of breath and excruciating air hunger, similar to the sensations experienced in drowning and near-drowning victims.

114. "Flash" or "acute" pulmonary edema refers to a phenomenon in which this entire process happens rapidly, e.g. over seconds to minutes, rather than over the course of hours or days. Symptoms of flash pulmonary edema in patients include cough, shortness of breath, air hunger, rapid breathing, sweating, falling levels of oxygen in the bloodstream, frothy sputum (sometimes pink or red due to blood) or fluid in the airway, and acute sensations of doom and/or drowning.[145]

115. I have reviewed the report of Dr. Edgar in this case and from that report I understand that pulmonary edema was present in 27 of 32 autopsies of prisoners executed by midazolam lethal injection. I also understand from Dr. Edgar's report that flash pulmonary edema developed during the three-drug lethal injection of these prisoners.

116. I further understand that defendants in this matter do not contest that pulmonary edema is a potential effect of the administration of midazolam at the dosage and concentration called for in Oklahoma's execution protocol.[146]

117. Based on my review of the expert report of Dr. Edgar, there is a high risk that prisoners executed using midazolam will develop flash pulmonary edema.

2. Midazolam Is Not an Analgesic or Sole Anesthetic Agent and Cannot Reliably Maintain a Prisoner in an Insensate and Anesthetics State During the Noxious Stimuli and Severe Suffering of Pulmonary Edema.

118. As stated above, Section VI.A.3, midazolam alone is not an analgesic and therefore does not reduce or diminish the experience of pain. Additionally, as explained in detail in Section

[145] *E.g.*, Gagne D. *Acute pulmonary edema*, *supra*.

[146] 11/12/2020 Crow Dep. tr. 199:18-200:7.

VI.A., midazolam does not reliably induce or maintain general anesthesia. Therefore, it is my opinion that midazolam used alone cannot prevent a prisoner from experiencing the severe suffering experienced during the pulmonary edema that is substantially likely to occur as a result of administration of midazolam as part of Oklahoma's three-drug lethal injection protocol.

119. It is therefore my further opinion that because midazolam is unlikely to render an inmate insensate to the severe suffering that will result from pulmonary edema there is a substantial and very high risk that the prisoners will experience severe suffering due to the sensations of suffocation, air hunger, and drowning they will experience as a result of the pulmonary edema caused by Oklahoma's three-drug lethal injection protocol.

C. Midazolam Cannot Reliably Maintain an Insensate or Anesthetic State During the Noxious Stimuli Caused by the Paralytic Agent.

120. The second drug in Oklahoma's lethal injection protocol is vecuronium bromide 100 milligrams of vecuronium bromide, which is approximately 10 times the usual clinical dose. The protocol does not specify the concentration used, but the dose is administered using two syringes each containing 50 mg.

1. The Vecuronium Bromide in Oklahoma's Lethal Injection Protocol Is Unnecessary to Effect or Speed Up Onset of Death.

121. Vecuronium bromide, the second drug in Oklahoma's three-drug protocol, a paralytic agent, serves no purpose in Oklahoma's lethal injection protocol other than to prevent the inmate from moving, communicating, or otherwise showing physical responses to the other drugs in the protocol.

122. I understand that Oklahoma's representative, Director Scott Crow, testified that the vecuronium bromide in Oklahoma's three-drug protocol is used to stop the prisoner's breathing, in addition to its use "to prevent involuntary movements on the part of the inmate and really, very

importantly, to maintain the dignity of the inmate through the process," and for the benefit of those watching the execution, so that the event is not disturbing.[147]

123. It is my opinion, however, that vecuronium bromide is unnecessary for producing death or for causing death more rapidly in a lethal injection protocol using potassium chloride, and specifically in Oklahoma's three-drug lethal injection protocol. This is because potassium chloride, in the dose given in Oklahoma's lethal injection protocol, will cause death quickly, and likely within minutes.[148] For example, one clinical study examined 30 heart surgery patients and found that cardiac arrest after concentrations of potassium chloride less than those used in Oklahoma's lethal injection protocol, occurred after an average of 44 seconds after administration.[149]

124. I understand that the potassium chloride called for in Oklahoma's lethal injection protocol is administered immediately after administration of the vecuronium bromide.[150] Thus there is only minimal, if any, delay between the onset of paralysis caused by administration of the vecuronium bromide, and the cardiac arrest caused by administration of potassium chloride.

125. The vecuronium bromide called for in Oklahoma's lethal injection protocol is therefore unnecessary for producing death or for causing death more rapidly in Oklahoma's three-drug lethal injection protocol.

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[147] 11/12/2020 Crow Dep. Tr. 97:15-98:2.

[148] *E.g.*, Jakobsen Ø et al. (2013) *Adenosine instead of supranormal potassium in cardioplegia: it is safe, efficient, and reduces the incidence of postoperative atrial fibrillation*. A randomized clinical trial. J Thorac Cardiovasc Surg. 145:812-818.

[149] Jakobsen Ø et al., *supra*.

[150] 11/12/2020 Crow Dep. Tr. 160:14-161:24.

2. Vecuronium Bromide Causes Extreme Suffering, Including Sensations of Air Hunger, Drowning, Panic, and Distress

126. Patients who have suffered intraoperative awareness while paralyzed including as a result of vecuronium, without being adequately sedated or anesthetized describe feelings of pain, extreme fear and panic, distress, sensations of suffocation, and inability to signal to providers with gestures or facial expressions that they are aware and suffering.[151] Indeed, such patients are commonly described as experiencing "outward calm and inner terror"; the outward appearance of the patient is serene because the paralysis, such as that produced by vecuronium and other paralytic agents, does not permit movement or changes of expression that otherwise clue an observer that the patient is in extreme terror and discomfort, even though beneath the serene exterior, the patient is fully aware and in agony.[152]

127. Because of the danger of suffering from neuromuscular blocking agents, the Institute for Safe Medication Practices (ISMP) includes this medication among its list of drugs which have a heightened risk of causing significant patient harm when used in error.[153] The Interdisciplinary Safe Medication Use Expert Committee of the United States Pharmacopeia (USP) also has recommended that hospitals, clinics, and other practice sites should institute special safeguards in the storage, labeling, and use of these agents and should include these safeguards in staff orientation and competency training. Additionally, the USP recommends that healthcare

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[151] *E.g.* Thomsen JL et al. (2015) *Awareness during emergence from anaesthesia: significance of neuromuscular monitoring in patients with butyrylcholinesterase deficiency*. Br J Anaesth. 2015 i78-i88; Chong ID et al., (2014) *Long-acting neuromuscular paralysis without concurrent sedation in emergency care*. Am J Emerg Med. 32:452-456; Moerman, awareness and recall during general anesthesia.

[152] *E.g.* Thomsen JL et al. *supra*; Moerman, awareness and recall during general anesthesia; Perry SW (1985) Psychological reactions to pancuronium bromide. Am J Psychiatry 142:1390-1391.

[153] Drugs, Access Medicine, McGraw-Hill Medical, Vecuronium, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com)

professionals should be on high alert (especially vigilant) whenever a neuromuscular-blocking agent (NMBA) is stocked, ordered, prepared, or administered.[154]

128. The Director of the ODOC testified that at least one purpose of the paralytic agent, vecuronium bromide, in Oklahoma's Execution Protocol, was to prevent movement of the inmate, and characterized this purpose as "preserving the dignity of the inmate".[155]

129. As unpleasant as these movements are to watch, however, they serve a critical purpose; they signal that the individual might not be unconscious, and should receive other types of drugs to reduce their awareness or pain and suffering. Masking such movements is maleficent to a dying individual because it prevents treatment of his or her suffering.

130. For these reasons, it is unethical to euthanize a companion animal using paralytic agents. The American Association of Veterinary Medicine forbids use of paralytic agents for euthanasia of companion animals as the "death drug", even though dying movements can be extremely distressing to owners. This is because a paralytic agent may mask the animal's suffering and hide indications that other drugs are needed.[156] The Humane Society of the United States, moreover, explicitly forbids the use of paralytics during euthanasia, because when an animal is paralyzed,

> while the animal appears to be unresponsive to sight and sound, he may still feel deep pain and may actually be experiencing fear and

[154] Drugs, Access Medicine, McGraw-Hill Medical, Vecuronium, Drugs | AccessMedicine | McGraw-Hill Medical (mhmedical.com).

[155] 11/12/2020 Dep. Tr. 101:4-17; Ty Alper, Anesthetizing the Public Conscience: Lethal Injection and Animal Euthanasia, 35 Fordham Urb. L.J. 817 (2008). Available at: https://ir.lawnet.fordham.edu/ulj/vol35/iss4/5.

[156] Leary S, Underwood W, Anthony R, et al. American Veterinary Medical Association Guidelines for the euthanasia of animals: 2013 edition. AVMA, Schaumburg IL.

> panic as he remains aware of his surroundings. For this reason, immobilizing agents are never appropriate for use in euthanasia.[157]

Thus, in many states, including Oklahoma, the use of paralytic drugs is explicitly forbidden in animal euthanasia as being inhumane.[158]

131. In the case of medical care for terminally ill human beings, it would be unthinkable as well as unethical to remove a patient from a ventilator, and then for appearances sake, paralyze them and leave them to suffocate in a lingering death; aware of what was happening to them, but unable to cry out for help. "This practice, whose primary purpose is to make the patient 'look' comfortable during the dying process, is not acceptable. Since these agents have no sedative or analgesic effects, their use cannot be justified as beneficial to the patient."[159] Professional guidelines in human medicine explicitly oppose such practice and state that it is both unethical and inhumane because it would mask signs of suffering that need treatment.[160]

132. Because the vecuronium bromide has no sedative or analgesic effects, and because the potassium chloride is administered immediately after the vecuronium bromide, it is my opinion that vecuronium bromide serves no other purpose than to prevent the prisoner from communicating, showing outward signs of distress or pain, or showing other reactions to Oklahoma's lethal injection protocol.

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[157] The Humane Society of the United States. Euthanasia Reference Manual, 2013. Available at: https://www.animalsheltering.org/sites/default/files/content/euthanasia-reference-manual.pdf Accessed Dec. 28, 2020.

[158] *See* OS §4-501 (2006).

[159] Truog RD, Campbell ML, Curtis JR, et al. *Recommendations for end-of-life care in the intensive care unit*: *A consensus statement by the American College of Critical Care Medicine*. Crit Care Med 2008; 36:953-63.

[160] Downar J, Delaney JW, Hawryluck L, Kenny L. *Guidelines for the withdrawal of life-sustaining measures*. Intens Care Med 2016; 42:1003-17; Truog RD, *supra.*

3. Midazolam Is Not an Analgesic or Sole Anesthetic Agent and Cannot Reliably Maintain a Prisoner in an Insensate and Anesthetics State During the Noxious Stimuli Caused by Vecuronium Bromide.

133. As explained above, Sections V.A and VI.1-3, midazolam alone is not an analgesic nor is it reliably used to induce or maintain general anesthesia. Midazolam alone therefore cannot reliably prevent a prisoner from experiencing the noxious stimuli and suffering caused by the paralytic agent, vecuronium bromide, called for by Oklahoma's execution protocol. For that reason, it is my opinion that a prisoner executed in accordance with Oklahoma's three-drug injection protocol is at substantial risk of experiencing severe suffering, from the administration of the vecuronium bromide required by the protocol.

D. Midazolam Cannot Reliably Maintain an Insensate or Anesthetic State During the Administration of Potassium Chloride.

1. Administration of Potassium Chloride Causes Severe Pain.

134. As noted above, injection of concentrations of potassium chloride of more than 80-100 mEq/L (milliequivalents per liter)[161] are known to cause severe pain. This pain does not stop after injection, because the injury to the lining of the blood vessels continues.

135. Given the fact that midazolam has no analgesic properties, and does not reliably render a patient in an insensate and analgesic state, and given that the anticipated dose and concentration of KCl to be administered in the Oklahoma protocol is almost certain to cause excruciating pain during injection, it is my expert opinion that there is a substantial risk the prisoner will be aware of the injection and experience severe pain as a result.

[161] The convention for dosage of mineral salts such as potassium chloride is in mEq, or in mEq per liter of whatever fluid in which they are diluted. A physician must always not only designate the total dose (mEq), but how to dilute the salt (mEq/L) because both affect the clinical effects. For context, a common dose range of potassium chloride (KCl) is 25 to 40 mEq, delivered in 1 liter of fluid (i.e. a concentration of 25 to 40 mEq/L) over 8 to 10 hours.

136. Additionally, because of the administration of vecuronium prior to the injection of potassium chloride, it is my expert opinion that there is a substantial risk that during executions under this protocol observers will not see any movements from the prisoner who is not under general anesthesia that they will recognize as indicating the prisoner is experiencing pain and suffering.

2. Midazolam Is Not an Analgesic or Sole Anesthetic Agent and Cannot Reliably Maintain a Prisoner in an Insensate and Anesthetic State During the Severe Pain Caused by Potassium Chloride.

137. As explained above, midazolam alone is not an analgesic nor is it reliably used to induce or maintain general anesthesia. Midazolam alone therefore cannot prevent a prisoner from experiencing the severe pain experienced during the administration of the potassium chloride called for by Oklahoma's execution protocol.

138. It is my further opinion that due to the use of vecuronium bromide in Oklahoma's three-drug execution protocol, which paralyzes the prisoner, making it impossible for the prisoner to move or communicate, it is unlikely that any outward signs of the pain experienced as a result of the administration of potassium chloride will be detectable by witnesses to the execution.

E. Oklahoma's "Consciousness Checks" Are Inadequate to Determine Whether a Prisoner Is Insensate to the Pain and Noxious Stimuli of the Second and Third Drugs of the Execution Protocol.

1. Execution Protocol Provisions Regarding "Consciousness Checks"

139. The Execution Protocol states:

> Throughout the procedure, the IV Team leader shall monitor the inmate's level of consciousness and electrocardiograph readings utilizing direct observation, audio equipment, camera and monitor as well as any other medically approved method(s) deemed

> necessary by the IV Team leader. The IV Team leader shall be responsible for monitoring the inmate's level of consciousness.[162]

140. With respect to administration of the three-drug lethal injection protocol set forth in Chart D of Attachment D to the Execution Protocol specifically, the execution protocol states, in relevant part:

> 3. When approximately five (5) minutes has elapsed since commencing the administration of the first chemical [midazolam], the IV Team leader, dressed in a manner to preserve their anonymity, shall enter into the room where the H Unit section chief and inmate are located to physically confirm the inmate is unconscious by using all necessary and medically-appropriate methods. The IV Team leader shall also confirm that the IV line remains affixed and functioning properly.
>
> 4. If confirmed the inmate is unconscious, an announcement will be made and the director will order the remaining chemicals be dispensed in the order they appear in the chart.
>
> . . .
>
> 6. If the inmate remains conscious after approximately five (5) minutes, the IV Team shall communicate this information to the director, along with all IV Team input. The director shall determine how to proceed or, if necessary, to start the procedure over at a later time or stop the execution. The director may order the curtains to the witness viewing room be closed, and if necessary, for witnesses to be removed from the facility.
>
> 7. If deemed appropriate, the director may instruct the Special Operations Team to administer additional doses of the chemical(s) followed by the heparin/saline flush.
>
> 8. Upon administering the chemical(s) and heparin/saline from a backup set, the IV Team shall confirm the inmate is unconscious by sight and sound, utilizing the audio equipment, camera and monitor. The IV Team leader shall again physically confirm the inmate is unconscious using proper

[162] Execution Protocol, at ¶ E.6.

> medical procedures and verbally advise the director of the same.

141. Oklahoma's execution protocol thus proscribes that if, after five minutes have elapsed from the time the prisoner is first administered midazolam, the IV Team Leader determines that the inmate remains conscious, the director determines, primarily on the basis of the recommendation of the IV team leader, whether the execution will proceed, and if so how.[163]

142. If the director determines to proceed with the execution, the director may instruct another member of the execution team, the Special Operations Team leader, to administer two additional syringes of midazolam, each containing a dose of 250 milligrams midazolam, for a total additional dose of 500 milligrams of midazolam, a combined total dose of 1000 milligrams of midazolam, followed by another 60 ml heparin/saline flush.[164]

143. After an unspecified amount of time, the IV Team will "confirm the inmate is unconscious by sight and sound, utilizing the audio equipment, camera and monitor."[165] The IV Team leader again "physically confirms the inmate is unconscious using proper medical procedures."[166]

144. The protocol does not specify what will occur if, at the second consciousness check, described in Paragraph H.8, it is determined that the prisoner still remains conscious.

145. The Execution Protocol therefore requires monitoring a prisoner's "consciousness" using electrocardiograph readings, direct observation, audio equipment, camera and monitor, and physical confirmation at specific points in the protocol. The Execution Protocol does not further

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[163] *Id.* at ¶ H.6; 12/17/2020 Crow Dep. Tr. at 68:14-21.

[164] *Id.* at ¶ H.7; 12/17/2020 Crow Dep. Tr. at 68:10.

[165] *Id.* at ¶ H.8; 12/17/2020 Crow Dep. Tr. at 160:9-10.

[166] *Id.* at ¶ H.8; 12/17/2020 Crow Dep. Tr. at 160:23-161:3.

describe the method, manner, interpretation, or outcome of these required assessments of "consciousness". The determination of whether any additional methods of assessing the prisoner's depth of anesthesia is left to the discretion of the IV team leader.

2. Testimony Regarding Oklahoma's Proposed "Consciousness Checks"

146. I have reviewed the testimony of Oklahoma Department of Corrections Director Scott Crow, and the anonymous declaration provided in response to written questions of Oklahoma's current IV team leader.

147. Director Crow testified in this matter that the IV team leader would perform a "sternum rub or an eyelid flick and, in some instances, observe the pupil of the eye to determine if the inmate is unconscious or not," and that "the method that they use to determine that is at the discretion of the IV team leader."[167]

148. Director Crow further testified that the phrase "all necessary and medically appropriate methods" means "[w]hat process or procedure the IV team leader chooses, based on his knowledge and experience, to determine if the inmate is conscious or not."[168] Director Crow also testified that the methods used are at the discretion of the IV team leader, and that he would consider the consciousness checks performed by the IV team leader to be inadequate "[o]nly in the instance if no methodology was used at all."[169]

149. Director Crow further testified that the IV team leader may use an EKG machine, oxygen sensor, and a blood pressure monitor to determine both whether the prisoner is unconscious and whether the prisoner is insensate to painful stimuli.[170] Director Crow explained that the IV

[167] 11/12/2020 Crow Dep. Tr. 152:15-153:6.

[168] 11/17/2020 Crow Dep. Tr. 162:3-9.

[169] 11/17/2020 Crow Dep. Tr. 162:3-18.

[170] 11/12/2020 Crow Dep. Tr. 154-166.

team leader has the ability to monitor the video camera and microphones placed in the room with the prisoner in order to monitor the prisoner using sight and sound.[171] The equipment for monitoring the video and microphones is located in the same place as the equipment for monitoring the prisoner's oxygen content, blood pressure, and EKG.[172] Director Crow testified that if the IV "team leader advises that the inmate is unconscious, and there's no other circumstances existing otherwise," he has no discretion to do other than dispense the remaining second and third chemicals in the protocol.[173]

150. When the second and third chemicals are dispensed, the IV team leader is located in a room with the monitoring equipment and manifold board, which is not in the same room as the prisoner.[174]

151. I have reviewed the declaration of the current IV team leader, given anonymously as Pat Doe, in response to written deposition questions.[175] The IV team leader testified in relevant part as follows:

> 2. What is the proposed methodology to be used to perform a consciousness check?
>
> **ANSWER: Do they respond to verbal stimulus? (ex: loud voice)**
> **Do they respond to painful stimulus? (ex: pinch the skin between two fingers, trap pinch, supraorbital pressure, sternal pressure or rub, applying pressure to nail bed)**
> **Check pupils for size, shape, and level of reactivity**
> **Check corneal reflex by using finger or cotton tip swab to brush upper eyelid or touch the cornea to observe if the eyelid closes.**

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[171] *Id.* 166:6-15.

[172] *Id.* 166:23-167:9.

[173] 11/17/2020 Crow Dep. Tr. 165:10-17.

[174] 11/17/2020 Crow Dep. Tr. 165:18-166:15.

[175] 01/06/2021 Pat Doe Decl. in Response to Written Dep. Questions.

. . .

2bii. What are the indicia that would confirm or establish that an individual is "unconscious" as that term is used in that paragraph?

ANSWER: Please refer to my previous answer to question 2 above.

2bvi. Please identify and describe in detail each of the "necessary and medically-appropriate methods" that could or might be used "to physically confirm the inmate is unconscious."

ANSWER: Please refer to my previous answer to question 2 above.

2bvi. Please identify and describe in detail each of the "necessary and medically-appropriate methods" that could or might be used "to physically confirm the inmate is unconscious."

ANSWER: Please refer to my previous answer to question 2 above.

. . .

2bvi2. Which of those methods do you intend to use in your role as IV Team Leader and why?

ANSWER: I intend to use all of the above mentioned methods. It is my duty to ensure to the best of my ability that the inmate is unconscious.

2bvi3. If you have not made that decision, when do you intend to do so?

ANSWER: I do feel these specific methods mentioned are subject to change based on protocols and training

. . .

2cv(1). How will you monitor the inmate's level of consciousness by utilizing "direct observation"?

ANSWER: You can monitor an inmate's level of consciousness by director observation based on verbal responses, motor responses, and previously discussed ocular movements and reflexes. If you would like more detail please refer to previous answers regarding these three topics.

. . .

2cvi(1). How will you monitor the inmate's level of consciousness by utilizing "audio equipment"?

ANSWER: Again, you would use audio equipment to monitor level of consciousness based on sound. Does the inmate respond verbally to a command? Are the verbal responses appropriate or inappropriate? The level of consciousness based on sound and or verbal responses are as follows: oriented and can talk and answer questions appropriately, confused but can still talk and speak coherently but not with appropriate orientation, inappropriate words and answers with inappropriate response to questioning, incomprehensible words and respond with unintelligible words or sounds, and none meaning no verbal response at all.

. . .

2cvii(l). How will you monitor the inmate's level of consciousness by utilizing "camera and monitor"?

ANSWER: you can monitor motor response with regards to level of consciousness using a camera. Do they obey commands, do they localize to pain, do they withdrawal to pain, do they exhibit decorticate posturing (flexion), do they exhibit decerebate posturing (extension), or do they have no motor movement at all.

. . .

2cviii. Please identify and describe in detail each of the "medically approved method(s)" that could or might be utilized to "monitor the inmate's level of consciousness."

ANSWER: I have answered the majority of this question through answering questions above. For description in detail please refers to previous answers on the appropriate topic. Again you can assess for response to verbal or painful stimuli. Check pupils and corneal reflex. Assess verbal responses as mentioned previously. Assess motor responses as mentioned previously. You can also monitor their vital signs and electrocardiograph, and use direct observation.

. . .

2cviii2(l). Which of those methods do you intend to use in your role as IV Team Leader and why?

> **ANSWER: I intend to use all of the above mentioned methods because I feel it is my duty to ensure to the best of my ability that the inmate is unconscious.**
>
> . . .
>
> 2cviii3. If you have not made those decisions, when do you intend to do so?
>
> **ANSWER: Based on the protocol I was given my intention is to use all of these available methods. I do feel these specific methods mentioned are subject to change based on protocols and training.**
>
> . . .
>
> 3. What are the criteria for determining whether any unanticipated difficulties during the execution will result in an interruption or delay of the execution procedure?
>
> **ANSWER: If I feel the patient is not adequately unconscious then this could result in an interruption or delay in the execution process. If at any point any team member determines that any part of the execution process is not going according to procedure, they shall advise the IV team leader who shall immediately notify the agency director. The agency director shall determine whether to go forward with the procedure, delay the procedure, or stop the execution.**

152. Oklahoma's "consciousness check" procedures are inadequate to determine whether a prisoner will be insensate and will remain in a state of general anesthesia in the presence of the pain and noxious stimuli of the second and third drugs of Oklahoma's execution protocol.

3. Oklahoma's Proposed "Consciousness Checks" Fail to Account for Varying Levels and Repetition and Sequence of Painful and Noxious Stimuli

153. As explained above, Section V.B.4, the American Society of Anesthesiologists considers changes in consciousness caused by medications on a continuum ranging from anxiolysis (anxiety relief) to general anesthesia. Assessment of the level of sedation is thus similarly determined using varying methods of stimulation, which increase in intensity commensurate with the corresponding depth of sedation of the subjects. Monitoring for depth of

anesthesia is thus done continuously and along a continuum because changes in mental status and response to noxious stimulus can be discussed as a continuum.

154. "In assessing the level of consciousness of the patient, it is necessary to determine the intensity of stimulation necessary to arouse a response and the quality of the response that is achieved."[176] Posner et al., explain that where a patient fails to "respond to voice or vigorous shaking, the examiner next provides a source of pain to arouse the patient. Several methods for providing a sufficiently painful stimulus to arouse the patient without causing tissue damage" are used, including "compression of the nail beds, the supraorbital ridge, or the temporomandibular joint."[177] "If there is no response to the stimulus, a more vigorous midline stimulus may be given by the sternal rub. By vigorously pressing the examiner's knuckles into the patient's sternum and rubbing up and down the chest, it is possible to create a sufficiently painful stimulus to arouse any subject who is not deeply comatose."[178]

155. Posner et al. further explain that the level of response at each sequential stimulus is

> important to the initial consideration of the depth of impairment of consciousness. In descending order of arousability, a sleepy patient who responds to being addressed verbally or light shaking, or one who responds verbally to more intense mechanical stimulation, is said to be lethargic or obtunded. A patient whose best response to deep pain is to attempt to push the examiner's arm away is considered to be stuporous, with localizing responses. Patients who make only nonspecific motor responses (wincing, restlessness, withdrawal reflexes) without a directed attempt to defend against the stimulus are considered to have a nonlocalizing response and are comatose. Patients who fail to respond at all are in the deepest stage of coma.[179]

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[176] Plum & Posner, *Diagnosis and Treatment of Stupor and Coma*, at p. 71.

[177] Plum and Posner, *supra*, at 71.

[178] Plum and Posner, *supra*, at 71-72.

[179] Plum and Posner, *supra*, at 72-73.

156. When anesthesia is initiated for surgery, as opposed to when consciousness is evaluated in potentially intoxicated, comatose, or injured patients, the depth of anesthesia and sedation can and should also be monitored in a sequential and stepwise manner, as a subject is under each of the sequential stages of anesthesia discussed above, Section V.B.3: induction, maintenance, and emergence.

157. As one of the leading references on anesthesia explains:

> When a hypnotic drug is administered to induce general anesthesia—usually as an intravenous bolus over a 5- to 10-second period—several physiologic signs are observed. If asked to count backwards from 100, the patient typically does not get beyond 85 to 90. This transition into unconsciousness can be followed easily by asking the patient to perform smooth pursuit of the anesthesiologist's finger. In smooth pursuit, the patient is instructed to move his or her eyes to track the position of the anesthesiologist's finger. As loss of consciousness ensues, the lateral excursions of the eyes during smooth pursuit decrease, nystagmus may appear, blinking increases, and the eyes fix abruptly in the midline.[180]

158. At the induction stage, in addition to assessing smooth pursuit, oculocephalic and corneal reflexes, as well as pupillary response are also used to assess depth of anesthesia. The "oculocephalic reflex and the corneal reflex are lost, but the pupillary response to light can remain intact. The patient typically becomes apneic [temporary cessation of breathing for short periods], atonic [lacking muscle tone], and unresponsive at the point when the oculocephalic reflex is lost."[181]

159. The oculocephalic reflex is considered a particularly important measure of a subject's depth of anesthesia. "The oculocephalic reflex is assessed by turning the patient's head from side to side, while lifting the eyelids. Before administration of the induction anesthetic, when

[180] Brown, *Monitoring*, *supra*, at 1280.

[181] *Id.*

the reflex is intact in a patient with no neurologic deficits, the eyes move in the direction opposite the motion of the head. When the reflex is lost, the eyes stay fixed in the midline."[182]

160. Another tool used to assess depth of anesthesia is corneal reflex.

> The corneal reflex has traditionally been assessed using a wisp of cotton at the corner of the eye to stroke the cornea. An easier way to assess the reflex is to allow a drop of sterile water to fall on the cornea. Using a drop of sterile water may be safer than using the wisp of cotton, because the former is less likely to cause a corneal abrasion. With either approach, the reflex is intact if the eyes blink consensually, is impaired if there is a blink in one eye and not the other, and is absent if there is no blink.[183]

161. Brown explains that the oculocephalic and corneal reflex are used together, along with the other indicia of loss of consciousness, including apnea, atonia, and lack of responsiveness, to infer that anesthesia has been successfully induced:

> Loss of the oculocephalic reflex suggests that the motor nuclei required for eye movements have been affected by the anesthetic. Similarly, loss of the corneal reflex suggests that the nuclei that control sensation and motor responses to sensation on the eyes and the face have also been affected. Because the loss of the oculocephalic and corneal reflexes occur concomitantly with the loss of responsiveness, the anesthesiologists can also infer that the loss of consciousness is due at least in part to the effects of the anesthetics on the nearby arousal centers.[184]

162. As described above, where a patient fails to "respond to voice or vigorous shaking, the examiner next provides a source of pain to arouse the patient."[185] This may include one or

[182] Brown, *Monitoring*, *supra*, at 1280.

[183] *Id.*

[184] *Id.*

[185] Plum and Posner, *supra*, at 71.

more of several methods, including "compression of the nail beds, the supraorbital ridge, or the temporomandibular joint."[186]

163. During maintenance of general anesthesia, a subject should be continuously monitored to ensure that the subject is not emerging from anesthesia prematurely. Several methods are used to continuously monitor a patient and ensure that they remain in general anesthesia, monitoring a patient's physiological signs, such as heart rate, and blood pressure, and may include EEG:

> EEG-based indices are among the most commonly used methods for tracking loss of consciousness induced by general anesthesia. With induction of general anesthesia, these indices usually change from high values that indicate the awake state to lower values that indicate states of sedation and unconsciousness. [187]

164. EEG indices cannot be used to predict exactly when a subject is certain to regain consciousness, but they are still commonly used to predict when a subject is likely to emerge from anesthesia:

> When delivery of the anesthetic drugs is decreased or terminated, the indices increase toward values that are consistent with the awake state. As the values of the indices increase, the patient is more likely to become conscious. In this way, the EEG-based indices can be used to monitor changes in anesthetic state during emergence from general anesthesia. [188]

165. Continuous physiological monitoring of subjects under anesthesia is particularly important:

> The state of the patient during emergence from general anesthesia can be tracked reliably by monitoring the patient's physiologic signs and performing neurologic examinations. Many of these physiologic changes relate to the return of brainstem function.

[186] Plum and Posner, *supra*, at 71.

[187] Brown, Monitoring, *supra* at 1280.

[188] Brown, Monitoring, *supra* at 1286.

> Therefore, by relating the physiologic signs and the findings from the neurologic examinations to the brainstem centers responsible for them, anesthesia providers can track the return of function to specific brainstem sites during emergence from general anesthesia.[189]

166. The reason for simultaneous, ongoing, and sequential use of assessment means is that any one of these may be inadequate on their own to determine whether the prisoner has reached a state of general anesthesia such that they will be insensate even in the presence of severe pain and noxious stimuli.

4. Oklahoma's Proposed "Consciousness Checks" Fail to Sufficiently and Continuously Monitor the Prisoner Throughout the Execution Process

a. Continuous Monitoring Is Essential

167. As discussed above, Section V.B.5, a number of protocols are routinely used simultaneously to continuously monitor whether an individual is and continues to be insensate and in a state of general anesthesia in a clinical setting. Appropriate assessment of induction and maintenance of general anesthesia includes continuous monitoring of physiologic parameters such as heart rate and blood pressure, assessment of both the oculocephalic and corneal reflexes, and *continuous* monitoring for signs of movement or vocalization, amongst other tools, such as EEG, discussed below.

168. Continuous monitoring is necessary because, as explained above, Section V.B.4, subjects may be aroused from certain depths of anesthesia only as a result of repeated or multiple stimuli. Thus, monitoring vital signs such as heart rate, blood pressure, and oxygen levels only at the point in time at which a subject is subjected to a single stimulus is not necessarily predictive of the subject's later response to a greater or repeated painful or noxious stimuli. Any test is just a

[189] Brown, Monitoring, *supra* at 1287-88.

measure at that particular moment and maintenance of anesthesia requires on going adjustment based on surgical noxious stimulus and the status of the prisoner or subject.

169. Additionally, not only physiological measurements are used to assess whether general anesthesia is maintained. Rather the neurological assessment methods described above, corneal reflex and oculocephalic reflex can be used, as are other physical signs of the loss of consciousness, such as swallowing, gagging, tearing, and grimacing. "These physiologic signs are often present in advance of the patient responding to any verbal commands."[190] Similarly, "[t]he corneal reflex typically returns before the oculocephalic reflex."[191] And "[t]he pupillary light reflex can remain intact even when the patient is profoundly unconscious under general anesthesia; therefore, the presence of the pupillary light reflex might not indicate a change in the level of consciousness while under general anesthesia." "Opening of the eyes is typically one of the last physiologic signs observed in patients emerging from general anesthesia. In particular, patients may respond reliably to verbal commands, have substantial return of motor functions, yet not necessarily open their eyes."[192] Anesthesiologists therefore use a combination of continuous monitoring and neurological assessment to determine anesthetic depth.

170. These physical and neurological observations require that the person assessing anesthesia be able to simultaneously monitor the physical state of the patient as well as their physiological measurements, such as heartrate and blood pressure.

171. From the testimony I have reviewed, it is unclear how the IV team leader would be able to monitor the prisoner's heart rate and blood pressure while simultaneously performing a

[190] Brown, *monitoring*, *supra* at 1286.

[191] *Id.*

[192] *Id.* at 1287-88.

physical assessment of the inmate in a room separate from the equipment used to monitor the inmate. It is therefore my opinion that Oklahoma's proposed "consciousness checks" are inadequate for at least this reason.

b. The Paralytic Agent Renders Most of the Assessment Tools Ineffective

172. Based on the above studies, standard anesthetic practice, and my own experience, it is my opinion that the only reliable method of determining that the prisoner is insensate to pain and suffering and will remain in a state of general anesthesia is to perform assessments of the oculocephalic and corneal reflex; to continuously monitor the prisoner's heart rate and blood pressure for signs of nociception; and to remove the use of the paralytic agent which has no purpose in the execution, in order to be able to sufficiently observe the patient for signs of movement or vocalization.

173. As explained above, Section V.A.2, the paralytic agent, vecuronium bromide, serves no purpose in Oklahoma's execution protocol other than to ensure that the prisoner cannot respond through movement, vocalization, or other communication, to any noxious stimuli. Because the paralytic agent prevents reliable monitoring of the prisoner for purposes of ensuring the prisoner is insensate and is in a state of general anesthesia throughout the execution, the paralytic should be removed.

174. Reports describing the only two midazolam executions conducted without the use of a paralytic also suggest that the paralytic masks movement, pain, and suffering. Dennis McGuire and Joseph Wood III were both executed with a protocol that used midazolam and hydromorphone without the use of a paralytic; in both executions, according to witnesses both prisoners heaved, moved, and gasped for breath during the execution.

175. Dennis McGuire was executed by the State of Ohio on January 16, 2014. An eyewitness offered this account: "The chemicals began flowing about 10:29 a.m., and for a while, McGuire was quiet, closing his eyes and turning his face up and away from his family. However, about 10:34 a.m., he began struggling. His body strained against the restraints around his body, and he repeatedly gasped for air, making snorting and choking sounds for about 10 minutes. His chest and stomach heaved; his left hand, which he had used minutes earlier to wave goodbye to his family, clenched in a fist."[193]

176. Similarly, a witness account of the execution of Joseph Wood III on July 23, 2014, by the State of Arizona, describes the prisoner gasping and heaving—by one eyewitness account, gasping more than 640 times.[194] The witness explains: "at 2:05, Wood's mouth opened. Three minutes later it opened again, and his chest moved as if he had burped. Then two minutes again, and again, the mouth open wider and wider. Then it didn't stop. He gulped like a fish on land. The movement was like a piston: The mouth opened, the chest rose, the stomach convulsed."[195]

177. These two executions using midazolam in the absence of a paralytic provide evidence that absent the use of a paralytic, a prisoner would be able to demonstrate movement and distress in response to painful or noxious stimuli. The removal of the paralytic would therefore allow for more comprehensive assessment and monitoring of the prisoner's depth of anesthesia.

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[193] Alan Johnson, *Inmate's death called 'horrific' under new, 2-drug execution*, The Columbus Dispatch (Jan. 16, 2014), https://www.dispatch.com/article/20140116/NEWS/301169709.

[194] Michael Kiefer, Reporter recounts nearly 2-hour execution, KVUE ABC (July 24, 2014), https://www.kvue.com/article/news/reporter-recounts-nearly-2-hour-execution/269-260131222.

[195] *Id.*

c. Oklahoma's Proposed "Consciousness Checks" Fail to Account for the Situation in Which a Prisoner Loses Adequate Anesthesia

178. Finally, nothing in the Execution Protocol provides for the situation in which a prisoner loses adequate depth of anesthesia to render the prisoner insensate to the second and third drugs of the execution protocol. It is my further opinion that in order to ensure the prisoner remains insensate and in order to ensure maintenance of general anesthesia, additional rescue medications should be available to maintain general anesthesia. Such drugs may include opioids such as fentanyl, propofol,

F. Oklahoma's Proposed "Consciousness Checks" Fail to Account for the Inadequacy of Certain Tests With Midazolam

179. Numerous studies have demonstrated that commonly used so called "consciousness checks" such as eyelash reflex, calling out to the patient, sternal rub, or other measures to evaluate depth of anesthesia, are ineffective for benzodiazepines generally, including midazolam.[196] Calling the patient's name, the eyelash reflex, or applying mild to moderately painful stimuli such as pinch and sternal rub similarly do not predict whether a patient is aware, nor whether they will have recall.[197]

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[196] *See, e.g.*, Gamble, et al., *Evaluation of midazolam as an intravenous induction agent*, Anaesthesia, 1981, Volume 36, pages 868-873, 872-73 ("The absence of the eyelash reflex was not a reliable end-point in that many patients in whom this was abolished opened their eyes on command . . . ."); *see also* sources cited *infra* footnote 197.

[197] Russell IF. *Comparison of wakefulness with two anaesthetic regimens. Total i.v. balanced anaesthesia*. Br J Anaesth 1986; 58:965-8; Russell IF. *The ability of bispectral index to detect intraoperative wakefulness during total intravenous anaesthesia compared with the isolated forearm technique*. Anaesthesia 2013; 68:502-11; Blackmon BB, Mahaffey JE, Baker JD. *Clinical comparison of midazolam hydrochloride and midazolam maleate for anesthesia induction*. Anesth Analg 1984; 63: 1116-20; Baker AB. *Induction of anesthesia with diazepam*. Anaesth 1969; 24:388-92.

180. Specifically, the eyelash reflex test is inadequate as a consciousness check for midazolam used alone as an anesthetic induction agent.[198] For example, Gamble et al. reported in one study that although many patients lost the "eyelash" reflex after administration of midazolam, they still responded to loud sounds and exhibited purposeful (conscious) movement with onset of the paralytic agent, indicating that they were not anesthetized or unconscious.[199] In a second study, Gamble et al., found that in 230 healthy patients given midazolam in combination with other drugs (i.e. not as a solo drug), that even when the anesthetist assessed the patient as being "asleep" after midazolam administration, many responded to moderate stimulation (endotracheal intubation) or surgical stimuli indicating that they were not.[200]

181. As explained above, quantification of MAC is another assessment tool used to determine depth of anesthesia.[201] Studies assessing MAC and midazolam show that certain of Oklahoma's previously used methods of testing "consciousness," such as the eyelash reflex and response to verbal stimulus, are inadequate to confirm whether a patient is insensate to painful or noxious stimuli, such as a surgical stimulus. These studies show that patients lose both their lid reflex and the response to verbal stimulus at 0.3 MAC.[202] But at even 0.7 MAC, 50% of people would still respond to a surgical stimulus. Therefore, the eyelash reflex and response to verbal

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[198] Gamble, et al., *supra*; Gear, et al., *Benzodiazepine mediated antagonism of opioid analgesia*, Pain 71 (1997) 25–29.

[199] Gamble et al., *supra*.

[200] Gamble JAS, Dundee JW, Kawar P. *Midazolam-an alternative to thiopentone?* Br J Anaesth 1980; 52:951p-952p.

[201] Avidan MS, Jacobsohn E, Glick D, Burnside B, Lini Z, Villafranca MS, Karl L, Kamal S, Torres B, O'Connor M, Evers AS, Gradwohl S, Lin N, Palanca BJ, Mashour GA. Prevention of Intraoperative Awareness in a High-Risk Surgical Population. New Eng J Med. 2011;365(7):591-600.

[202] Avidan, et al., *supra.*

stimulus are wholly inadequate to determine whether an individual will be insensate to a painful stimulus equal to that of a surgical stimulus or to the pain caused by the third drug in Oklahoma's protocol or to the suffering caused by the vecuronium bromide or the pulmonary edema.

182. The pupillary assessment identified by the IV Team Leader is also unreliable. Visual assessment with the naked eye is unreliable– automated quantification using infrared pupillometry is now possible "Visual assessment of pupillary activity with the naked eye have poor test retest and interrater reliability."[203] "Eight to eighteen percent of normal individuals have anisocoria greater than 0.4 millimeters," meaning they have one pupil that is larger than the other. This makes it difficult to assess pupillary activity.

183. Thus, in addition to the corneal reflex, anesthesiologists also test the oculocephalic reflex, which is considered an important tool for assessing deeper levels of anesthesia. "Because the oculomotor circuitry enfolds and surrounds most of the arousal system, this part of the examination is particularly informative."[204] [The oculocephalic reflex … in patients with impaired consciousness this should be the main thing or something like that. -doll's eyes]. Some individuals can also lose the oculocephalic reflex as a result of wearing contact lenses.[205]

1. Oklahoma's Proposed "Consciousness Checks" Overlook Current Relevant Techniques for Assessing Depth of Anesthesia

184. The IV team leader fails to mention oculocephalic reflex and continuous monitoring of physiological responses.

[203] Plum & Posner, *supra.*

[204] Plum & Posner, *supra.*

[205] Plum & Posner, *supra.*

185. Additionally, EEG is now typically used to continuously monitor a subject's depth of anesthesia, especially where a patient will be paralyzed during the procedure, which renders many assessment tools ineffective.

> EEG-based indices are among the most commonly used methods for tracking loss of consciousness induced by general anesthesia. With induction of general anesthesia, these indices usually change from high values that indicate the awake state to lower values that indicate states of sedation and unconsciousness.[206]

186. It is unclear why the IV team leader references decerebrate and decorticate posturing, but … many patients with decerebrate/decorticate posturing are actually "conscious". If the IV team leader's belief is that these movements indicate a lack of consciousness, studies have shown that is incorrect. Decerebrate/decorticate posturing is consistent with the prisoner still experiencing pain, but the absence of these movements do not indicate that the individual is not experiencing. Decerebrate/decorticate posturing is usually produced in response to a painful stimulus. intends to rely on these movements as indicia that the patient is "unconscious," that is incorrect.

2. Oklahoma's Proposed "Consciousness Checks" Fail to Address the Inadequacy of the Same Techniques in Prior Executions

187. I understand that the consciousness checks set forth in the current protocol and described by witness testimony are the same that were used unsuccessfully in the execution of Clayton Lockett.[207] Similar techniques for checking consciousness used in the execution of Joseph Wood also suggest that Oklahoma's proposed "consciousness checks" are inadequate.

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[206] Brown, *Monitoring*, *supra* at 1280.

[207] *See* Oklahoma Dep't of Public Safety, *The Execution of Clayton D. Lockett Case Number 14-0189SI, Executive Summary*, at 11 (Exhibit 70); May 27, 2014 Physician Interview Transcript, Department of Public Safety Investigation.

188. The investigation of the Oklahoma Department of Safety provides the following timeline:

- 6:23 p.m.: "The full dose of midazolam and an appropriate saline flush were administered. A DOC employee began to keep time on a stopwatch."
- 6:30 p.m.: "The signal was given that five minutes had elapsed and the physician determined Lockett was conscious. DOC personnel began to keep additional time on a stopwatch."
- 6:33 p.m.: "The signal was given that two minutes had elapsed and the physician determined Lockett was unconscious. [The warden] signaled for the execution to continue. The full dose of vecuronium bromide, an appropriate saline flush, and a majority of the potassium chloride were administered."
- 6:33-6:42 p.m.: "Lockett began to move and make sounds on the execution table."
- The execution was then stopped; Lockett subsequently died in the execution chamber. [208]

189. The doctor responsible for determining that Lockett was first conscious and then later determined to be unconscious described the methods used to assess Lockett's depth of anesthesia as follows: "Oh, I blow in his eyes, I'll rub on his sternum, and I pinch him. I check his, his eye movement to see if there is any eye movement."[209]

190. Joseph Wood's execution provides another example of the failure of the "consciousness checks" similar to those proposed here. A reporter eyewitness describes the following:

- At 1:54 p.m., "[t]he drugs had already begun to flow through the IVs. . . . Four minutes into the procedure, the doctor appeared on the other side of the window.

[208] Oklahoma Dep't of Public Safety, *The Execution of Clayton D. Lockett Case Number 14-0189SI, Executive Summary*, at 11 (Exhibit 70).

[209] May 27, 2014, Physician Interview Transcript, Department of Public Safety Investigation.

> He checked Wood's eyes and pulse and then said over the microphone, 'It is confirmed that he is sedated.'"[210]

- "[A]t 2:05, Wood's mouth opened. Three minutes later it opened again, and his chest moved as if he had burped. Then two minutes again, and again, the mouth open wider and wider. Then it didn't stop. He gulped like a fish on land. The movement was like a piston: The mouth opened, the chest rose, the stomach convulsed."[211]

- "And when the doctor came in to check on his consciousness and turned on the microphone to announce that Wood was still sedated, we could hear the sound he was making: a snoring, sucking, similar to when a swimming-pool filter starts taking in air, a louder noise than I can imitate, though I have tried."[212]

191. The testimony and eyewitness accounts of the Lockett and Wood executions, which appear to have used similar "consciousness checks" to those currently proposed by Oklahoma in its current protocol, confirm that these consciousness checks were inadequate to determine that the prisoners were not insensate and were not in a state of general anesthesia—as that term is defined by the American Society of Anesthesiologists—in the presence of painful or noxious stimuli.

192. Additionally, even if the protocol required more rigorous assessments of depth of anesthesia, proper assessment of anesthetic depth and application of the tools and techniques described above requires extensive training, which is not required by the Execution Protocol. Thus, even if every one of the above tools were required by the protocol, it is unclear from the protocol that they would implemented appropriately because the protocol does not require the IV team leader—or any other member of any execution team—to have any training in assessing depth of anesthesia.

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[210] Michael Kiefer, *Reporter recounts nearly 2-hour execution*, KVUE ABC (July 24, 2014), https://www.kvue.com/article/news/reporter-recounts-nearly-2-hour-execution/269-260131222.

[211] *Id.*

[212] *Id.*

3. Oklahoma's Training and Experience Requirements Are Inadequate to Ensure the IV Team Leader Is Qualified to Assess Depth of Anesthesia

a. Assessment of Whether a Prisoner Is Insensate and in a State of General Anesthesia Requires Significant Training and Experience

193. "The examiner must be conversant with the meaning of the signs elicited in the examination, so that decisions . . . can be made quickly and accurately."[213] The drugs and protocol being used will dictate the level of training and experience required to properly assess depth of anesthesia. The use of midazolam alone to induce anesthesia at dosages that have not been studied is a protocol that necessitates the highest level of proficiency in performing assessment of depth of anesthesia.

194. The requirements set forth in the Execution Protocol for the IV team leader are not sufficient to ensure that someone has the training and experience necessary to sufficiently assess and monitor the anesthetic depth of the prisoner in order to maintain the prisoner under general anesthesia so as to render the prisoner insensate to severe pain and noxious stimuli.

b. The Execution Protocol Does Not Require Any Training or Experience Related to Assessment of Depth of Anesthesia

195. The Execution Protocol indicates that "[t]he IV Team shall consist of a team leader and member(s) of any one or more of the following": physician(s) physician's assistant(s), nurse(s), emergency medical technician(s), paramedic(s), military corpsman, or "other certified or licensed personnel including those trained in the United States military."[214]

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[213] Plum & Posner, *supra*.

[214] *Id.* at ¶ IV.C.1.

196. The IV team leader is solely responsible for performing all of the "consciousness checks" called for in the Execution Protocol.[215] And none of the operations team members or H-unit-team members are required to have any medical training.[216]

197. The IV team leader is also essentially solely responsible for determining when an execution should be called off due to complications related to administration of the execution chemicals and consciousness checks. While the IV team leader testified that "[t]he agency director shall determine whether to go forward with the procedure, delay the procedure, or stop the execution,"[217] the agency director testified that he would base his decision "to go forward with the procedure, delay the procedure, or stop the execution," primarily on the advice of the IV team leader.[218] Thus the IV team leader has a central role both in determining the anesthetic depth of the prisoner, and determining the appropriate recourse in the event of an unexpected development or problem during the execution.

198. The Protocol does not indicate whether or what, if any, training, experience, or credentials, the IV Team leader shall have prior to joining the IV team, other than having one of the qualifications set forth above, including that the IV team leader may be an "other certified or licensed personnel" of an unspecified type.[219]

199. It is my opinion that the requirements set forth in the Execution Protocol for the IV team leader are insufficient to ensure that the IV team leader will be able to sufficiently assess and monitor the anesthetic depth of the prisoner in order to maintain the prisoner under general

[215] 11/17/20 Crow Dep. Tr. 160:23-161:3.

[216] Execution Protocol, at ¶ IV. C.

[217] Pat Doe deposition

[218] 11/12/2020 Crow Dep. Tr. 160:23-161:3

[219] *Id.*

anesthesia so as to render the prisoner insensate to severe pain and noxious stimuli. To be clear, it is not my opinion that a nurse, an emergency medical technician, or a paramedic, such as those called for in the execution protocol, would not be acceptable if those persons had sufficient training and experience; but the fact that someone is certified as a medical doctor, nurse, or other qualification listed in the Execution Protocol is not in and of itself sufficient to ensure they have the skills necessary to conduct the assessments of anesthetic depth set forth above.

4. Prisoners Executed Using Oklahoma's Execution Protocol Face a Substantial Risk of Severe Pain and Suffering Due to the Inadequacy of Oklahoma's Proposed "Consciousness Checks" and Lack of Appropriate Training and Experience Requirements

200. The "consciousness checks" contemplated by Oklahoma's written execution protocol are deficient for numerous reasons. First, the execution protocol only purports to measure "consciousness" and not depth of anesthesia. Determination of "consciousness" alone is inadequate, for the reasons set forth throughout this report, but in particular in Section V.B.1, for determining whether a prisoner is insensate to pain and suffering and in a state of general anesthesia.

201. Second, the written protocol's requirements for sufficient training and experience of the individual responsible for the "consciousness checks" are inadequate. For the reasons discussed above, the requirements for the IV team leader are inadequate to ensure that the IV team leader will be capable of assessing adequate levels of anesthesia under any drug protocol.

202. Third, Oklahoma's three-drug lethal injection protocol is using midazolam as the only drug for sedation and anesthesia, which is not how midazolam is typically used, as set out in detail in sections VI.A.1-2. Because midazolam is not typically used for the purpose for which it is being used in the execution protocol, the assessment of the prisoner's depth of anesthesia

requires the highest levels of skill to ensure that the prisoner is maintained at an adequate anesthetic depth and in an insensate state.

203. Fourth, the only required assessments of "consciousness" required by the written protocol—electrocardiograph readings, direct observation, audio equipment, camera and monitor, and physical confirmation—are inadequate on their own to ensure that the prisoner is insensate and maintained in a general state of anesthesia. Moreover, the protocol doesn't specify the manner and appropriate application of each of these assessments for evaluating the depth of anesthesia and sedation. Instead, determination of the medically appropriate consciousness checks is entirely at the discretion of the IV team leader. The actual means that will be used to assess the "consciousness" or depth of anesthesia of the prisoner are entirely unspecified. To the extent they are specified, they are inadequate.

204. Fifth, the consciousness checks identified by the current IV team leader are similarly unspecified, as they are at the discretion of whomever is serving as the IV team leader at the time of a given execution. Moreover, the current team leader acknowledged that even as to the current protocol and team leader, the consciousness checks are expected to change, based on the protocol and training.[220] To the extent they are specified, they are inadequate for all of the same reasons set forth directly above.

205. Sixth, the execution protocol makes no provisions for the situation in which a prisoner is deemed to be adequately "unconscious" but is later aroused from sedation by an adequately severe painful or noxious stimulus. That this is possible is demonstrated by the Lockett, Wood, and Maguire executions. Yet, despite awareness of at least the Lockett execution,

[220] Doe Decl. at 4.

Oklahoma's execution protocol fails to provide for appropriate means of maintain sufficient anesthetic depth to maintain the prisoner in an insensate state.

206. For all of the above reasons, it is my opinion that prisoners executed using Oklahoma's three-drug lethal injection protocol face a substantial risk of experiencing pain and suffering.

G. Additional Deficiencies in the Training and Experience Requirements Set out in Oklahoma's Execution Protocol Increase the Likelihood that a Prisoner Will Experience Severe Pain and Suffering

207. As the Lockett execution shows, IV site preparation and placement is crucial to proper administration of the execution chemicals. Proper IV site preparation and placement requires proper training and experience.

208. Despite awareness of at least the Lockett execution, Oklahoma's Execution Protocol lacks adequate training and qualification requirements to ensure proper iv site preparation and placement.

209. Oklahoma's Execution Protocol provides that the Special Operations Team "[i]mplements the protocols associated with the preparation and administration of the chemicals for the execution."[221] The Special Operations Team consists of a Team leader, a Recorder, and three additional team members.[222] The Execution Protocol does not indicate whether or what, if any, training, experience, or credentials the members of the Special Operations Team are required to have.[223]

[221] Execution Protocol, at ¶ IV. B.3.

[222] *Id.*

[223] *See id.*

210. The additional IV team members, while identified as having one of the certifications identified above, e.g., nurse, emergency medical technician, similarly set out no additional requirements with respect to experience involving I preparation and placement of the IV line.

211. Additionally, after joining the IV team, the IV team members are required only to "conduct a minimum of two training sessions with multiple scenarios within seven days prior to the scheduled execution, with the H Unit Section Teams.[224]

212. Given the complexity of the application and analysis of the assessment of anesthetic depth and continuous monitoring, and the necessity of proper preparation and placement of the IV line in order to ensure proper administration of the execution chemicals, the training requirements for the H-Unit, Special Operations team members, and the additional IV team members are inadequate because they are not related to the medical skills required by these execution team members. Similarly, in light of the lack of training and experience requirements, the complexity of the assessments involved, and the possible contingencies that may be necessary in response to the many unexpected problems that can arise during an execution, two days of training with the IV team leader may also be inadequate.

VII. CONCLUSION

213. In conclusion,

- It is my opinion that prisoners executed in accordance with Oklahoma's Execution Protocol, OSP Policy No. OP-040301, face a substantial risk of experiencing severe pain and suffering as a result of the Protocol's execution drugs and procedures.

- It is also my opinion that midazolam, administered alone, cannot reliably render and maintain a subject in an insensate state and induce and maintain anesthesia, or block nociception (perception or transmission of a painful stimulus). It is

[224] *Id.* at ¶ V.C.

widely accepted that midazolam, a sedative, should not be used as the only drug to induce and maintain anesthesia or render a patient insensate to pain.[225] If midazolam is used as an induction agent, it is typically used in combination with other drugs for rendering a patient insensate and for inducing anesthesia.[226] It is neither approved nor used for maintaining general anesthesia without other drugs.[227]

- It is my further opinion that there is significant individual variation in response to midazolam, which can require a significant range in dosing to achieve similar results between individuals.[228] This variation is based on a number of factors, including, individual genetic differences, pharmacokinetics (the way the body metabolizes a drug), interaction with other drugs, and existing medical conditions.[229] This is one reason why midazolam is, as a general matter, not appropriate for use as a sole induction agent for general anesthesia and is not used as a sole agent for maintenance of general anesthesia.

- It is also my opinion that midazolam is commonly referred to and understood as having a "ceiling effect."[230] This means that independent of any individual variation in responsiveness, additional amounts of the drug, even at much higher doses, will not produce an equally greater response in a subject or have a proportionally greater effect.

- It is my further opinion that midazolam does not reliably render and maintain a subject in an insensate state and induce and maintain general anesthesia, or block nociception, in the presence of severe noxious stimuli, such as intense pain, or a sensation of suffocation or paralysis.

- It is also my opinion that the three-drug protocol, including midazolam, in the dose and concentration called for in the protocol, leads to flash pulmonary

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[225] *E.g.*, Package Insert, Pfizer, Midazolam Injection, USP, at Indications and Usage.

[226] E.g., *id.*

[227] *E.g.*, *id.*; Amrein & Hetzel, *Pharmacology of Dormicum® (midazolam) and Anexate® (flumazenil)*, Acta Anasesthesiol Scand 1990: 34, Supplementum 92:6-15.

[228] *See, e.g.*, *id.* at Warnings ("Midazolam must never be used without individualization of dosage."); *E.g.*, Amrein & Hetzel, *supra* at 8 ("patients may react differently to equal doses of a given BZD or ***vice versa*** different doses may be needed to achieve the same effect in different patients").

[229] *E.g.*, Amrein & Hetzel, *supra*.

[230] *E.g.*, Amrein & Hetzel, *supra* at 6 ("a ceiling effect is observed after maximal doses of midazolam").

edema, a condition involving severe suffering.[231] During flash pulmonary edema fluid floods the lungs, causing sensations of air hunger, drowning, and/or suffocation. Because midazolam does not reliably render a subject insensate, and maintain general anesthesia, or block nociception, in the presence of severe noxious stimuli, it is my further opinion that midazolam cannot reliably render and maintain a subject in an insensate state during the severe suffering caused by flash pulmonary edema.

- It is my further opinion that midazolam is unlikely to render and maintain a subject in an insensate state and induce and maintain general anesthesia in the presence of the pain or suffering caused by vecuronium bromide and potassium chloride, the second and third drugs in ODOC's three-drug protocol, following midazolam administration. In particular, vecuronium bromide administered alone results in a slow death by asphyxiation caused by paralysis, which prevents a subject's ability to communicate and move. Potassium chloride administered alone produces cardiac arrest and a sensation of burning and intense pain as it circulates through the body. Midazolam is unlikely to render and maintain a subject in an insensate state and will not maintain general anesthesia, or block nociception, in the presence of the noxious stimuli caused by the effects of vecuronium bromide and the severe pain caused by potassium chloride.

- It is my opinion that the "consciousness check" method described in Paragraphs H.3 and H.8 of Attachment D to the Execution Protocol is not sufficient to ensure a subject is insensate to the noxious stimuli of pulmonary edema, effects of vecuronium bromide, and potassium chloride.

- It is my further opinion that if the first and third drugs in ODOC's three-drug protocol, midazolam and potassium chloride, were administered to a subject without the second drug in the three-drug protocol, vecuronium bromide, a subject would be able to communicate or otherwise demonstrate through physical or other responses, their experience of flash pulmonary edema as described above, including the sensations of shortness of breath and excruciating air hunger, similar to a sensation of drowning. The subject would also be able to communicate or otherwise demonstrate through physical or other responses, their experience of burning, and intense pain as the potassium chloride circulated through their body. This is based on my opinion above that midazolam administered alone is unlikely to render a subject insensate and maintain unconsciousness in the presence of severe noxious stimuli, such as intense pain. Without administration of vecuronium bromide the subject would be able to communicate the sensations experienced or at a minimum show

[231] *See, e.g.*, Ikram, et al., Intravascular infusion of acid promotes intrapulmonary inducible nitric oxide synthase activity and impairs blood oxygenation in rats. Critical Care Medicine 2003; 31: 1454-1460 and Am J Respir Crit Care Med 1999: 159: 397-402.

physical response resulting from administration of potassium chloride and the midazolam itself.

- Additionally, it is my opinion that the personnel requirements, training requirements, "consciousness check" provisions, intravenous ("IV") site preparation and establishment provisions, and the equipment provisions of the Execution Protocol, as described below, create or contribute to a risk that a prisoner executed in accordance with the Execution Protocol will experience serious harm and severe pain and suffering. These requirements and provisions relate to medical decisions necessitated by the Execution Protocol procedures and potential medical outcomes that will result from the Execution Protocol procedures. Additional provisions or changes to the existing procedural, training, or equipment requirements would mitigate the risk that a prisoner executed in accordance with the Execution Protocol will experience serious harm and severe pain and suffering.

214. I reserve the right to respond to any opinions offered by Defendants' experts in this matter.

I declare that I have examined this report and all statements contained herein, and to the best of my knowledge and belief, they are true, correct and complete.

/s/ Michael L. Weinberger
Michael L. Weinberger

January 11, 2021
DATE

EXHIBIT A

Michael L. Weinberger

Department of Anesthesiology - Division of Pain Medicine
Columbia University Medical Center

622 West 168th Street
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New York, NY 10032
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mlw45@columbia.edu

EDUCATION

Columbia University	**M.D.**	**New York, NY**	**1983**
Clark University	**B.A.**	**Worcester, MA**	**1979**

POSTDOCTORAL TRAINING

Date	Field	Institution
1983–1984	Internship Internal Medicine	St. Vincent's Hospital/Medical Center New York, NY
1984–1986	Resident Internal Medicine	St. Vincent's Hospital/Medical Center New York, NY
1986–1989	Resident Anesthesiology	Columbia-Presbyterian Medical Center New York, NY
1989-1990	Fellow, Pain Management	Memorial Sloan Kettering Cancer Ctr. New York, NY

LICENSED PHYSICIAN

YEAR	LICENSE NUMBER	STATE OF ISSUE
1984	165889	New York
1992 -1999	58457	New Jersey

BOARD CERTIFICATION

American Board of Internal Medicine	1986
American Board of Anesthesiology	1990
American Board of Anesthesiology (Pain Medicine)	1993, 2003.2014
American Board of Hospice and Palliative Medicine	2006
American Board of Anesthesiology (Hospice and Palliative Medicine)	2012

POSITIONS & APPOINTMENTS

Date	Title and Department	Institution
1986–1989	*Visiting Fellow* Anesthesiology	Columbia University New York, NY
1989–1990	*Clinical Assistant Anesthesiologist* Department of Anesthesiology Critical Care Medicine	Memorial Hospital for Cancer and Allied Diseases New York, NY
1989–1990	*Clinical Assistant* Department of Anesthesiology Critical Care Medicine	Memorial Sloan Kettering New York, NY
1989–1990	*Instructor in Anesthesiology* Department of Anesthesiology	Cornell University Medical College New York, NY
1990–1992	*Assistant Attending Anesthesiologist* Department of Anesthesiology Critical Care Medicine	Memorial Hospital for Cancer and Allied Diseases New York, NY
1990–1992	*Assistant Clinical Member* Department of Anesthesiology Critical Care Medicine	Memorial Sloan Kettering New York, NY
1990–1992	*Assistant Professor of Anesthesiology* Department of Anesthesiology	Cornell University Medical College New York, NY
1992–1998	*Assistant Attending Anesthesiologist*	Overlook Hospital Summit, NJ
1998–2001	*Assistant Attending Anesthesiologist*	St. Vincent's Hospital/Medical Center New York, NY
1999–2001	*Assistant Professor of Anesthesiology*	New York Medical College Valhalla, NY
1999–2001	*Fellowship Director* Pain Management	St. Vincent's Hospital/Medical Center New York, NY
2000–2001	*Medical Director*	University Pain Center New York, NY
2001–	*Medical Director* The Pain Management Center, Department of Anesthesiology	Columbia University Medical Center (New York Presbyterian Hospital) New York, NY

2001– 2013	*Fellowship Director* Division of Pain Medicine	Columbia University Medical Center (New York Presbyterian Hospital) New York, NY
2001–2003	Assistant Clinical Professor Anesthesiology	Columbia University Medical Center New York, NY
2003 - 2013	Associate Clinical Professor of Anesthesiology	Columbia University Medical Center
2007-2010	Medical Director Palliative Medicine	Columbia University Medical Center
2013	Associate Professor of Anesthesiology at CUMC	Columbia University Medical Center

SCIENTIFIC & MEDICAL SOCIETIES

Eastern Pain Association,
International Association for the Study of Pain
American Society of Anesthesiologists
American Society of Regional Anesthesia and Pain Medicine
New York State Society of Anesthesiologists
International Spinal Injection Society

COMMITTEES

NYPH Spine Governance Committee	2018 -
ASRA Guidelines Committee	2019 -
ASRA Research Committee	2019 -
NYSSA Pain Committee	2018 -
NYPH Pain Committee	2018 -
Pain Committee, American Society Anesthesiology	2017 -
NYPH Opioid Work Group	2016
President, Eastern Pain Association	2010- 2012
Board of Directors Executive Committee, Eastern Pain Association	2008 -
Palliative Medicine Special Interest Group, American Pain Society	2005-2012
Chairperson, Palliative Medicine SIG, American Pain Society	2006-2012
Board of Directors, Eastern Pain Association	2005-2014
Program Committee (GRIPE), New York Pain Group	2005-2014
Program Committee, Eastern Pain Society Annual Meeting	2004
Oncology Operations Council, New York Presbyterian Hospital	2004- 2012
Pain Medicine Subcommittee, Pharmacy and Therapeutics Committee, CUMC	2004- 2008
New York State Pain Initiative	2003- 2006
President, New York State Pain Initiative	2004- 2006

Faculty Practice Organization, New York Presbyterian Hospital	2003-2005
Ethics Committee, NYPH / Columbia University Medical Center, CUMC	20001-2014
Department of Anesthesiology Resident Education Committee, CUMC	2001-
201Department of Anesthesiology Service Committee, CUMC	2001-
JCAHO Pain Task Force, New York Presbyterian Hospital(NYPH)	2001
Palliative Care Quality Improvement Committee, NYPH / CUMC	2001
JCAHO Pain Task Force, St. Vincent's Hospital and Medical Center	2000
Geriatric Pain Treatment Committee, St. Vincent's Hospital and Medical Center	1998–1999
Executive Committee, NJSSA	1995-1998
Vice Chairman, Pain Subcommittee, NJSSA	1995–1997
Pain Task Force, Overlook Hospital	1992–1994
Post Graduate Assembly, Social Activity Committee**,** NYSSA	1990–1992
House Staff Education and Surveillance, St. Vincent's Hospital	1983–1986

PAPER

1. Dodds TM, Stone JG, Coromilas J, Weinberger M, Levy DG. Prophylactic nitroglycerin infusion during noncardiac surgery does not reduce perioperative ischemia. Anesth Analg 1993; 76:705-13
2. Barakat R, Sararian K, Sheperd G, **Weinberger M**, Hoskins WJ. Allergy to Latex Surgical Gloves: An unfamiliar cause of intraoperative anaphylaxis. Gynecol Oncol 1992; 6: 381-83

BOOK CHAPTERS

Cervical Disc Disorders in Head, Face and Neck Pain: Science, Evaluation and Management. N. Mehta, G Maloney, D Bana, SJ Scrivani. Publisher WJ Wiley Blackwell 2009

Complex Regional Pain Syndrome in Merritt's Neurology Twelfth Edition Lewis P Roland, Timothy A Pedley Publisher Wolters Kluwer Lippincot Williams & Wilkins Philadelphia 2010

Complex Regional Pain Syndrome in Merritt's Neurology Thirteenth Edition Publisher Wolters Kluwer Lippincot Williams & Wilkins Philadelphia 2015

LECTURES

1. Hospice Saint John. "Spinal Opioids." Wilkes-Barre, PA; 09/13/90.
2. Surgical Grand Rounds, Memorial Sloan Kettering Cancer Center. "The Use of Calcium Channel and Beta Receptor Blocking Agents in the Perioperative Period." New York, NY; 02/27/91.
3. Memorial Sloan Kettering Cancer Center. Current Concepts in Cancer and Acute Pain Management. "Spinal Opioids." New York, NY; 12/06/91
4. Surgical Grand Rounds, Memorial Sloan Kettering Cancer Center. "The Role of Epidural Narcotics in Postoperative Analgesia." New York, NY; 01/15/92.
5. Why Do We Care: Pain and Symptom Control, Psychiatric Issues and Ethical Dilemmas in the Care of Patients with Cancer, Memorial Sloan Kettering Cancer

Center. "Epidural Infusions and Anesthetic Blocks in Cancer Pain Patients." New York, NY; 04/04/92.

6. Orthopedic Grand Rounds, St. Vincent's Hospital and Medical Center. "Neural Blockade and Low Back Pain." New York, NY; 1998.
7. Division of Geriatric Medicine, St. Vincent's Hospital and Medical Center. "Geriatric Pain Management." New York, NY; 1999.
8. General Surgery Grand Rounds, St. Vincent's Hospital and Medical Center. "Cancer Pain Management." New York, NY; 1999.
9. Palliative Care Grand Rounds, St. Vincent's Hospital and Medical Center. "Multi-disciplinary Pain Management." New York, NY; 1999
10. 53rd Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologists. "Challenges in Acute Pain Management." New York, NY; 12/12/99
11. 53rd Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Chronic Pain." New York, NY; 12/13/99.
12. Internal Medicine Grand Rounds, St. Vincent's Hospital and Medical Center. "Neural Blockade and Low Back Pain." New York, NY; 08/2000.
13. Memorial Sloan Kettering Cancer Center. "Neural Blockade and Low Back Pain." New York, NY; 11/2000.
14. 54th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Management of Chronic Pain: Evaluation and Treatment." New York, NY; 12/09/00.
15. 54th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Challenges in Acute Pain Management." New York, NY; 12/09/00.
16. Grand Rounds, Department of Anesthesiology, University of Colorado Health Sciences. "Current Concepts in Low Back Pain." 03/2001.
17. Grand Rounds, Department of Medicine, Bergen Regional Medical Center. "Pain Management in the Elderly." Paramus, NJ; 04/2001.
18. Grand Rounds, Pain Management Divisions of Mt. Sinai, NYU, and Hospital for Joint Disease. "Current Concepts in Low Back Pain." New York, NY; 03/2001.
19. 55th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management." 12/2001.
20. Pain Management Rounds, New York University Medical Center. "Back Pain: Perspectives from the Pain Clinic." New York, NY; 2002.
21. Grand Rounds, Department of Neurological Surgery, Columbia Division of New York Presbyterian Hospital. New York, NY; 2002.
22. Anesthesia Pain Group, Memorial Sloan Kettering Cancer Center. "Back Pain: Perspectives from the Pain Clinic." New York, NY; 04/2002.
23. Integrative Pain Medicine. "Review of Invasive Procedures and Devices in Pain Medicine." New York, NY; 05/16/02.
24. Grand Rounds, Division of Hematology Oncology, Columbia Division of New York Presbyterian Hospital. "Evolving Concepts in Neuraxial Analgesia." New York, NY; 06/2002.
25. Grand Rounds, New York Orthopaedic Hospital. "Update on Opioid Analgesia." New York, NY; 10/2002.

26. Grand Rounds, Division of Hematology Oncology, Columbia Division of New York Presbyterian Hospital. "Terminal Sedation." New York, NY; 10/2002.
27. 56th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management." New York, NY; 12/2002.
28. Anesthesia in the 21st Century. "Back Pain; Perspectives from the Pain Clinic." 02/2003.
29. Anesthesia in the 21st Century. "Current Concepts in Opioid Analgesia." 02/2003
30. Grand Rounds, Department of Anesthesiology, New York University. "Low Back Pain." New York, NY; 03/2003.
31. Integrative Pain Medicine. "Review of Invasive Procedures and Devices in Pain Medicine." 05/16/03.
32. Pain Update, Hackensack University Medical Center. "Update on Opioid Mechanisms." Hackensack, NJ; 10/2003.
33. Grand Rounds, Internal Medicine, St. Vincent's Hospital of Bridgeport. "Back Pain." Bridgeport, CT; 11/2004.
34. Anesthesia in the 21st Century. "Intrathecal Therapies: Indications and Limitations." Miami, FL; 02/2004.
35. Anesthesia in the 21st Century. "Pain and Palliative Care: New Drugs, New Techniques." Miami, FL; 02/2004.
36. Grand Rounds, Department of Medicine, Allen Pavilion, New York Presbyterian Hospital. "Pain and Palliative Care: New Drugs, New Techniques." New York, NY; 04/15/04.
37. Integrative Pain Medicine, Third Annual Conference, Columbia University Medical Center. "Review of Invasive Procedures and Devices in Pain Medicine." New York, NY; 04/16/04.
38. Care of the Dying Patient: The Nursing Perspective, Columbia University Medical Center. "Pain Management and Palliative Sedation in Palliative Care." New York, NY; 05/05/04.
39. 2004 Annual Update and Intensive Review of Internal Medicine, Columbia University Medical Center and Beth Israel Deaconess Medical Center. "Chronic Pain Management." New York, NY; 08/06/04.
40. Department of Emergency Medicine, New York Presbyterian Hospital-Columbia Campus. "Interventional Approaches to Back Pain." New York, NY; 09/29/04.
41. 58th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management." New York, NY; 12/2004.
42. Quad Institutional Pain Medicine Grand Rounds, Department of Anesthesiology, New York Presbyterian Hospital-Cornell Campus. "Opioids in Chronic Pain." New York, NY; 01/12/05.
43. Integrative Pain Medicine 4th Annual Conference, Columbia University Medical Center. "Update on Opioid Therapy: From Poppies to Long-Acting Opioids." New York, NY; 04/15/05.

44. Integrative Pain Medicine 4th Annual Conference, Columbia University Medical Center. "Review of Invasive Procedures and Devices in Pain Medicine." New York, NY; 04/16/05.
45. CA-1 Resident Lecture, Department of Anesthesiology, Columbia University Medical Center. "Chronic Pain Management." New York, NY; 04/27/05.
46. Department of Internal Medicine, Allen Pavilion, New York Presbyterian Hospital. "Geriatric Pain Management and Palliative Care." New York, NY 05/11/05
47. Department of Internal Medicine Noon Conference, New York Presbyterian Hospital-Columbia Campus. "Palliative Care." New York, NY; 06/03/05.
48. CA-2 Resident Lecture, Department of Anesthesiology, Columbia University Medical Center. "Palliative Care." New York, NY; 06/28/05.
49. 2005 Annual Update and Intensive Review of Internal Medicine. Columbia University Medical Center and Beth Israel Deaconess Medical Center. "Chronic Pain Management." New York, NY; 08/05/05.
50. Quad-Institutional Pain Medicine Grand Rounds, Weill Medical College of Cornell University. "Opioids in Chronic Pain." New York, NY; 08/17/05.
51. 59th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management Workshop." New York, NY; 12/2005.
52. 59th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Update on Interventional Pain Management." New York, NY; 12/2005.
53. Doctor of Nursing Practice Program, School of Nursing, Columbia University. "Chronic Pain Management." New York, NY; 01/25/06.
54. Integrative Pain Medicine. "Review of Invasive Procedures and Devises in Pain Medicine." New York, NY; 04/07/06.
55. American Pain Society Annual Meeting 2006. "Palliative Medicine in Acute Care and Outpatient Settings." San Antonio, TX; 05/04/06.
56. 2006 Annual Update and Intensive Review of Internal Medicine. Columbia University Medical Center and Beth Israel Deaconess Medical Center. "Chronic Pain Management." New York, NY; 08/05/06.
57. New York University (NYU) Current Review of Pain Medicine. "New Spinal Drugs and Systems." New York, NY; 11/17/06.
58. 60th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management Workshop." New York, NY; 12/2006.
59. 60th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Update on Interventional Pain Management." New York, NY; 12/2006.
60. Chinese Association for the Study of Pain. "Intrathecal Therapies" Beijing 10/13/07
61. "Pain Medicine in the United States" Ruijin Hospital. Shanghai Jiaotong University School of Medicine Shanghai, China 10/17/07

62. Back Pain : "Perspectives from an Interventionalist and the Intersection with radiology" Dept of Radiology, Neuroradiology Section. Columbia University Medical Center New York, NY 10/24/07
63. Grand Rounds. "Pain Management Review and Update". Creedmor Psychiatric Center Queens Village, N.Y. 11/8/07
64. 61th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Update on Interventional Pain Management." New York, NY; 12/2007.
65. 61th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management Workshop." New York, NY; 12/2007.
66. Pain Medicine Grand Rounds, Department of Anesthesiology, New York Presbyterian Hospital-Cornell Campus. "Post Graduate Training Education: A Roadmap for Success." New York, NY; 05/21/08.
67. 2008 Annual Update and Intensive Review of Internal Medicine. Columbia University Medical Center. "Pain Medicine and Palliative Medicine Update." New York, NY; 07/25/08
68. 62th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Update on Interventional Pain Management." New York, NY; 12/2008..
69. 62th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. Scientific Panel "Intradiscal techniques" New York, NY; 12/2008.
70. 63th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Update on Interventional Pain Management." New York, NY; 12/2009.
71. 64th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Update on Interventional Pain Management." New York, NY; 12/2010.
72. 64th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Prevention of Injuries with Spinal Interventions in Pain Management Facet Radiofrequency Rhizotomy." New York, NY; 12/2010.
73. New York Chapter of Geriatric Care Managers "Pain Management in the Elderly" New York, NY; 6/1/2011
74. Annual Update and Intensive Review of Internal Medicine 2011
"Principles of Pain Medicine" New York, NY; 7/25/2011
75. Grand Rounds, Department of Anesthesiology Columbia University
"Lessons from Closed Claims Data: Spinal Cord Injury and Interventional Pain"
New York, NY; 8/4/2011
76. Current Concepts in Opiod Therapy for Chronic Intractable Pain
"Current Concept in Management of Chronic Non-Malignant Pain and the Role of Opioids" New York, NY; 11/12/2011
77. 65th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologist. "Interventional Pain Management Update" New York; 12/2011

78. Grand Rounds, Department of Anesthesiology, University of Vermont. "Palliative Medicine and the Anesthesiologist", Burlington, VT; 3/1/2012
79. Eastern Pain Association Annual Meeting "MILD – "Minimally Invasive Lumbar Decompression" New York, NY 12/8/2012
80. 66th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologists. "Update on Interventional Pain" New York, NY 12/14/2012
81. 66th Annual New York Post Graduate Assembly (PGA), New York State Society of Anesthesiologists. "Common Interventional Pain Procedures: What is the Evidence" New York, NY 12/16/2012
82. 67th Annual New York Post Graduate Assembly, New York State Society of Anesthesiologists " Update on Interventional Pain, Hypotensive Headache".New York, NY 12/13/2013
83 Grand Rounds George Washington University Department of Anesthesiology. "Opioids and Chronic Pain : An Update" 1/22/2014
84. Lyme Disease Association/ Columbia Lyme Conference. "Opioids and Chronic Pain:" 5/4/2014 Providence Rhode Island
85. Grand Rounds, Department of Anesthesiology Columbia University. "Opioids for Chronic Pain" 7/31/2014
86. 68th Annual New York Post Graduate Assembly, New York State Society of Anesthesiologists " Update on Interventional Pain, New Techniques in Spinal Cord Stimulation". New York, NY 12/4/2014
87. 69th Annual Post Graduate Assembly, New York State Society of Anesthesiologists "Pain Medicine from the last year: Articles you may have missed" 12/11/2015
88. 70th Annual Post Graduate Assembly, New York State Society of Anesthesiologists "Pain Medicine Update: New Developments in Spinal Cord Stimulation" 12/13/2016
89. Combined Grand Rounds, Department of Anesthesiology and Orthopedics. Columbia University. Morbidity and Mortality "Pain Management Challenges in Chronic Opioid Use/Abuser" 9/14/2017
90. Essentials in Pain Management: Update 2017. "Opioid Prescribing: Avoiding the Pitfalls and Employing Safe Strategies" New York Presbyterian Hospital/Columbia University Medical Center 11/14/2017
91. Pancreas Symposium 2018: Current Controversies in Benign Pancreatic Disease. "Pain Management in Chronic Pancreatitis" New York Presbyterian Hospital/Columbia University Medical Center 3/22/2018
92. Essentials on Pain Medicine: Update 2018. Peripheral Nerve Stimulation New York Presbyterian Hospital/Columbia University Medical Center 11/13/2018
93. 72th Annual Post Graduate Assembly, New York State Society of Anesthesiologists "Pain Medicine Update: Peripheral Nerve Stimulation and New Developments in Spinal Cord Stimulation" New York, NY 12/7/2018
94. Essentials on Pain Medicine: Update 2019 Radiofrequency Ablation:Emerging Targets, Utility in Joint Pain New York Presbyterian Hospital/Columbia University Medical Center 11/12/2019
95. Essentials on Pain Medicine: Update 2018. Neuropathic Pain Presbyterian Hospital/Columbia University Medical Center 11/12/2019

96. East Hill Rehabilitation Society. Cervical Epidurals: Injection Therapies for Cervical Radicular Pain. Closter, NJ. 12/3/2019

VISITING PROFESSORSHIP

"Palliative Medicine and the Anesthesiologist" 2/2012
Grand Rounds, Department of Anesthesiology
University of Vermont College of Medicine

"Current Concepts in Low Back Pain" 3/2001
Grand Rounds, Department of Anesthesiology
University of Colorado Health Sciences

"Opioids in Pain Medicine" 1/2014
Grand Rounds, Department of Anesthesiology
George Washington University

Pain in Chronic Pancreatitis 4/2018
Gastroenterology Rounds, Department of Medicine
Mt Sinai Medical Center. NY, NY

GRANTS & AWARDS

Oak Foundation - Palliative Medicine
Samuels Foundation Grant – Palliative Care Program 2008
Boston Scientific – Pain Fellowship – PI 2011-2013

EXHIBIT C

THE AMERICAN BOARD OF ANESTHESIOLOGY, INC.
A Member Board of the American Board of Medical Specialties

4208 Six Forks Road, Suite 900, Raleigh, North Carolina 27609-5735
Phone: (866) 999-7501 Fax: (866) 999-7503 Website: www.theABA.org

February 7, 2011

ABA IDN: 3582-0634

Richard Irvin Cook, MD
5841 South Maryland Avenue, # Mc4028
Chicago, IL 60637-1447

Dear Dr. Cook:

In response to your letter dated December 13, 2010, the American Board of Anesthesiology (ABA) writes to inform you that your testimony regarding lethal injection must be strictly limited to a discussion of the therapeutic or medical effects of certain anesthetics. In providing such testimony, you must be careful not to provide any testimony or opinions that would constitute a recommendation, endorsement or preferential suggestion of any form with respect to one anesthetic over another as such drugs may be used in connection with lethal injection.

The American Medical Association's Capital Punishment policy (which is enclosed) states that "in the case where the method of execution is lethal injection, the following actions by the physician would also constitute physician participation in execution: selecting injection sites; starting intravenous lines as a port for a lethal injection device; prescribing, preparing, administering, or supervising injection drugs or their doses or types; inspecting, testing, or maintaining lethal injection devices; and consulting with or supervising lethal injection personnel". Thus, in order to conform to the ABA's Professional Standing Policy, your testimony must be strictly limited to the medical effect of certain anesthetics on the patient's body, and not include or extend to a recommendation, endorsement, or preferential suggestion or statement that may lead one to conclude that one form of anesthetic is preferred over another in the context of a discussion regarding lethal injection.

Please note that this response is being provided to you alone and is not intended or being offered by the ABA for reliance by other third parties. We hope that this is responsive to your inquiry and we appreciate your caution in this area.

Sincerely,

David L. Brown, MD
ABA Secretary

DLB/lp

Enclosure

E-2.06 Capital Punishment

An individual's opinion on capital punishment is the personal moral decision of the individual. A physician, as a member of a profession dedicated to preserving life when there is hope of doing so, should not be a participant in a legally authorized execution. Physician participation in execution is defined generally as actions which would fall into one or more of the following categories: (1) an action which would directly cause the death of the condemned; (2) an action which would assist, supervise, or contribute to the ability of another individual to directly cause the death of the condemned; (3) an action which could automatically cause an execution to be carried out on a condemned prisoner.

Physician participation in an execution includes, but is not limited to, the following actions: prescribing or administering tranquilizers and other psychotropic agents and medications that are part of the execution procedure; monitoring vital signs on site or remotely (including monitoring electrocardiograms); attending or observing an execution as a physician; and rendering of technical advice regarding execution.

In the case where the method of execution is lethal injection, the following actions by the physician would also constitute physician participation in execution: selecting injection sites; starting intravenous lines as a port for a lethal injection device; prescribing, preparing, administering, or supervising injection drugs or their doses or types; inspecting, testing, or maintaining lethal injection devices; and consulting with or supervising lethal injection personnel.

The following actions do not constitute physician participation in execution: (1) testifying as to medical history and diagnoses or mental state as they relate to competence to stand trial, testifying as to relevant medical evidence during trial, testifying as to medical aspects of aggravating or mitigating circumstances during the penalty phase of a capital case, or testifying as to medical diagnoses as they relate to the legal assessment of competence for execution; (2) certifying death, provided that the condemned has been declared dead by another person; (3) witnessing an execution in a totally nonprofessional capacity; (4) witnessing an execution at the specific voluntary request of the condemned person, provided that the physician observes the execution in a nonprofessional capacity; and (5) relieving the acute suffering of a condemned person while awaiting execution, including providing tranquilizers at the specific voluntary request of the condemned person to help relieve pain or anxiety in anticipation of the execution.

Physicians should not determine legal competence to be executed. A physician's medical opinion should be merely one aspect of the information taken into account by a legal decision maker such as a judge or hearing officer. When a condemned prisoner has been declared incompetent to be executed, physicians should not treat the prisoner for the purpose of restoring competence unless a commutation order is issued before treatment begins. The task of re-evaluating the prisoner should be performed by an independent physician examiner. If the incompetent prisoner is undergoing extreme suffering as a result of psychosis or any other illness, medical intervention intended to mitigate the level of suffering is ethically permissible. No physician should be compelled to participate in the process of establishing a prisoner's competence or be involved with treatment of an

incompetent, condemned prisoner if such activity is contrary to the physician's personal beliefs. Under those circumstances, physicians should be permitted to transfer care of the prisoner to another physician.

Organ donation by condemned prisoners is permissible only if (1) the decision to donate was made before the prisoner's conviction, (2) the donated tissue is harvested after the prisoner has been pronounced dead and the body removed from the death chamber, and (3) physicians do not provide advice on modifying the method of execution for any individual to facilitate donation. (I) Issued July 1980. Updated June 1994 based on the report "Physician Participation in Capital Punishment," adopted December 1992, (*JAMA. 1993; 270: 365-368*); updated June 1996 based on the report "Physician Participation in Capital Punishment: Evaluations of Prisoner Competence to be Executed; Treatment to Restore Competence to be Executed," adopted in June 1995; Updated December 1999; and Updated June 2000 based on the report "Defining Physician Participation in State Executions," adopted June 1998.

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